



This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

### Usage guidelines

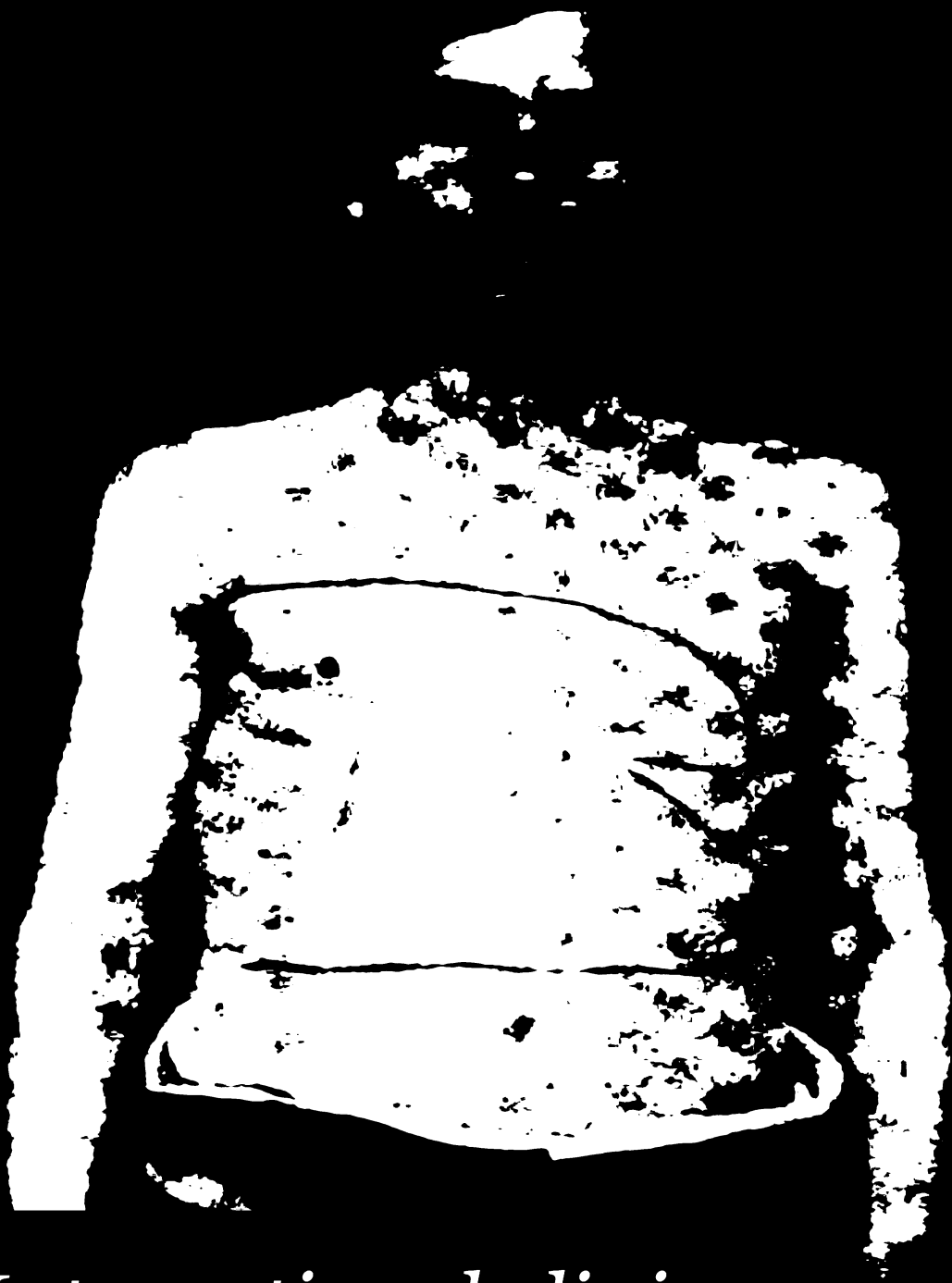
Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

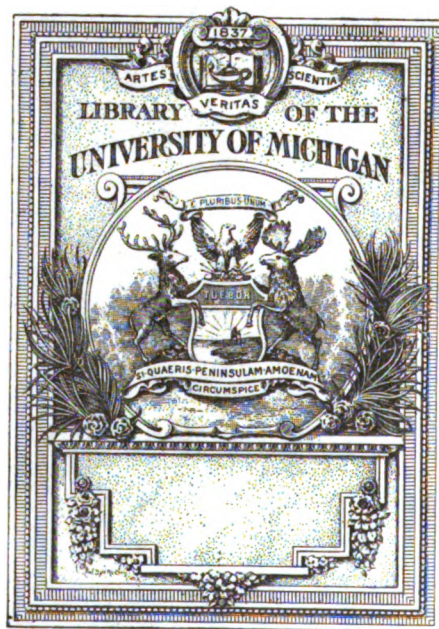
- + *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.
- + *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.
- + *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.
- + *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

### About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at <http://books.google.com/>



*International clinics*



610.5

T6

C64













# INTERNATIONAL CLINICS:

A QUARTERLY OF CLINICAL LECTURES

ON

MEDICINE, NEUROLOGY, SURGERY, GYNÆCOLOGY,  
OBSTETRICS, OPHTHALMOLOGY,  
LARYNGOLOGY, PHARYNGOLOGY, RHINOLOGY,  
OTOLOGY, AND DERMATOLOGY,

*AND SPECIALLY PREPARED ARTICLES ON TREATMENT AND DRUGS.*

BY PROFESSORS AND LECTURERS IN THE LEADING  
MEDICAL COLLEGES OF THE UNITED STATES,  
GERMANY, AUSTRIA, FRANCE, GREAT  
BRITAIN, AND CANADA.

EDITED BY

JUDSON DALAND, M.D. (UNIV. OF PENNA.), PHILADELPHIA,  
Instructor in Clinical Medicine and Lecturer on Physical Diagnosis in the University of Pennsylvania;  
Assistant Physician to the Hospital of the University of Pennsylvania; Professor of  
Clinical Medicine in the Philadelphia Polyclinic; Fellow of the  
College of Physicians of Philadelphia.

J. MITCHELL BRUCE, M.D., F.R.C.P., LONDON, ENGLAND,  
Physician to and Lecturer on the Principles and Practice of Medicine in the Charing Cross Hospital.

DAVID W. FINLAY, M.D., F.R.C.P., ABERDEEN, SCOTLAND,  
Professor of Practice of Medicine in the University of Aberdeen; Physician to and Lecturer on Clinical  
Medicine in the Aberdeen Royal Infirmary; Consulting Physician to the Royal  
Hospital for Diseases of the Chest, London.

VOLUME I. EIGHTH SERIES. 1898.

PHILADELPHIA:

J. B. LIPPINCOTT COMPANY.  
1898.

**Copyright, 1898, by J. B. LIPPINCOTT COMPANY.**

**PRINTED BY J. B. LIPPINCOTT COMPANY. PHILADELPHIA, U.S.A.**

# CONTRIBUTORS TO VOLUME I.

## (EIGHTH SERIES.)

---

**Anderson, T. McCall, M.D.**, Professor of Clinical Medicine in the University of Glasgow; Physician to the Western Infirmary, etc., Glasgow, Scotland.

**Ballantyne, J. W., M.D., F.R.C.P.E., F.R.S.E.**, Lecturer on Midwifery and Diseases of Women in the School of the Royal Colleges, Edinburgh; Examiner in Midwifery in the University of Aberdeen, etc., Scotland.

**Biss, Cecil Y., M.A., M.D. (Cantab.), F.R.C.P.**, Physician to the Out-patients' Department and Lecturer on Pharmacology and Therapeutics in the Middlesex Hospital Medical College, London, England.

**Bramwell, Byrom, M.D., F.R.C.P. (Edin.), F.R.S. (Edin.)**, Physician to the Royal Infirmary, Edinburgh; Lecturer on the Principles and Practice of Medicine in the School of Medicine, etc., Edinburgh, Scotland.

**Bridge, Norman, M.D.**, Professor of Clinical Medicine and Physical Diagnosis in Rush Medical College, Chicago, Illinois.

**Cantrell, J. Abbott, M.D.**, Professor of Diseases of the Skin in the Philadelphia Polyclinic and College for Graduates in Medicine; Dermatologist to the Philadelphia Hospital and to the Frederick Douglass Memorial Hospital.

**Coe, Henry C., M.D.**, Professor of Clinical Gynecology in the Bellevue Hospital Medical College, New York.

**Cumston, Charles Greene, B.M.S., M.D.**, Assistant Professor of Surgical Pathology, Faculty of Medicine, Tuft's College; Fellow of the American Association of Obstetricians and Gynecologists; Corresponding Member of the Association of Genito-Urinary Surgeons of France, of the Pathological Society of Brussels, of the Electro-Therapeutical Society of France, etc.

**Dabney, Samuel G., M.D.**, Professor of Physiology and Clinical Lecturer on Diseases of the Eye, Ear, Nose, and Throat in the Hospital College of Medicine, etc., Louisville, Kentucky.

**Daland, Judson, M.D. (University of Pennsylvania)**, Philadelphia. Instructor in Clinical Medicine and Lecturer on Physical Diagnosis in the University of Pennsylvania; Assistant Physician to the Hospital of the University of Pennsylvania; Professor of Clinical Medicine in the Philadelphia Polyclinic; Fellow of the College of Physicians of Philadelphia, Pennsylvania.

**Davis, N. S., Jr., A.M., M.D.**, Professor of Medicine and of Clinical Medicine in the Northwestern University Medical School, Chicago, Illinois.

**Hayem, Professor**, Clinical Professor of Medicine in the Paris Faculty of Medicine, and Visiting Physician to St. Anthony's Hospital, Paris, France.

iii



**Hirschfelder, J. O., M.D.**, Professor of Clinical Medicine in Cooper Medical College; Visiting Physician to the City and County Hospital, San Francisco, California.

**Hutchins, M. B., M.D.**, Clinical Lecturer on Dermatology and Syphilology, Atlanta Medical College, Atlanta, Georgia.

**Ingals, E. Fletcher, M.D.**, Professor of Laryngology in Rush Medical College; Professor of Diseases of the Throat and Chest, Woman's Medical College; Physician and Surgeon to the Central Free Dispensary for Diseases of the Chest, Throat, and Nasal Cavities, Chicago, Illinois.

**Jaccoud, Professor**, Visiting Physician to Hospital Pitié, and Professor of Medicine in the Faculty of Medicine, Paris, France.

**James, Alexander, M.D., F.R.C.P. (Edin.)**, Physician to the Edinburgh Royal Infirmary, Edinburgh, Scotland.

**Love, I. N., M.D.**, Professor of Clinical Medicine and Diseases of Children in Marion-Sims College of Medicine, and Professor of the Theory and Practice of Medicine in the Woman's Medical College, St. Louis.

**Marcus, Herman D., M.D.**, Late Lecturer on *Materia Medica* and Therapeutics in the Medico-Chirurgical College, Philadelphia, Pennsylvania.

**Marfan, Professor, M.D.**, Professor Agrégé in the Medical Faculty of Paris, France.

**Mathews, Joseph M., M.D.**, Professor of Surgery and Clinical Lecturer on Diseases of the Rectum in the Kentucky School of Medicine; Rectologist to the Kentucky School of Medicine Hospital and the Louisville City Hospital, etc., Louisville, Kentucky.

**Maylard, A. Ernest, B.S., M.B. (Lond.)**, Surgeon to the Victoria Infirmary, Glasgow, Scotland.

**Montgomery, E. E., M.D.**, Professor of Clinical Gynecology in the Jefferson Medical College; Gynecologist to Jefferson and St. Joseph's Hospitals; President of the Philadelphia Obstetrical Society; Ex-President of the Pennsylvania State Medical Society, etc., Philadelphia, Pennsylvania.

**Moore, William Oliver, M.D. (Columbia)**, Professor of Diseases of the Eye and Ear in the New York Post-Graduate Medical School and Hospital; Attending Ophthalmic Surgeon to the Orphans' Home and Asylum of the Protestant Episcopal Church, New York; Visiting Surgeon to the Post-Graduate Hospital, etc., New York.

**Mundé, Paul F., M.D., LL.D.**, Professor of Gynecology at the New York Polyclinic and at Dartmouth College; Gynecologist to Mt. Sinai Hospital, New York City, New York.

**Oliver, Thomas, M.A., M.D., F.R.C.P.**, Physician to the Royal Infirmary, Newcastle-upon-Tyne, England.

**Pinard, A., M.D.**, Clinical Professor of Obstetrics in the Faculty of Medicine, Paris.

**Pooley, Thomas R., M.D.**, Professor of Ophthalmology in the New York Polyclinic; Surgeon-in-Chief to the New Amsterdam Eye and Ear Hospital, New York City, New York.

**Randall, B. Alexander, M.D.**, Clinical Professor of Ear-Diseases, University of Pennsylvania; Professor of Otology, Philadelphia Polyclinic; Eye and Ear Surgeon to the Methodist and Children's Hospitals, etc.

**Roberts, W. O., M.D.**, Professor of Surgery and of Clinical Surgery in the University of Louisville; Surgeon to the Louisville City Hospital, etc., Louisville, Kentucky.

**Robin, Albert, M.D.**, Professor Agrégé in the Medical Hospital of Paris; Member of the Academy of Medicine, etc., France.

**Steell, Graham, M.D. (Edin.), F.R.C.P. (Lond.)**, Physician to the Manchester Royal Infirmary, and Lecturer on Diseases of the Heart and in Clinical Medicine, Owens College, Manchester, England.

**Stockton, Charles G., M.D.**, Professor of the Principles and Practice of Medicine and Clinical Medicine in the Medical Department of the University of Buffalo; Attending Physician to the Buffalo General Hospital, Buffalo, New York.

**Thomas, J. D., M.D.**, Professor of Genito-Urinary Diseases in the Medical Department of the Western Pennsylvania University; Surgeon and Genito-Urinary Surgeon to South Side Hospital, Pittsburg, Pennsylvania.

**Thomson, Robert Stevenson, M.D., B.Sc.**, Visiting Physician to the City of Glasgow Small-pox Hospital, Scotland.

**Tirard, Nestor, M.D. (Lond.), F.R.C.P.**, Physician to the King's College Hospital and to the Evelina Hospital for Sick Children, London, England.

**Todd, J. S., M.D.**, Professor of Therapeutics in the Atlanta Medical College; First Assistant Surgeon-General of the United Confederate Veterans, etc., Atlanta, Georgia.

**Von Leyden, Professor E.**, Professor of Special Pathology and Therapeutics and Director of the First Medical Clinic in the University of Berlin, Germany.

**Walker, H. O., M.D.**, Professor of Rectal Surgery, Genito-Urinary Diseases, and Clinical Surgery, in the Detroit College of Medicine, Detroit, Michigan.

**Wathen, William H., M.D., LL.D.**, Professor of Obstetrics, Abdominal Surgery, and Gynæcology in the Kentucky School of Medicine; Fellow of the American Gynæcological Society and of the Southern Surgical and Gynæcological Society; Gynæcologist to the Kentucky School of Medicine Hospital and the Louisville City Hospital, etc., Louisville, Kentucky.



# CONTENTS OF VOLUME I.

## (EIGHTH SERIES.)

---

### Drugs and Remedial Agents.

	PAGE
<b>CONTRAINDICATIONS TO THE USE OF SALICYLATE OF SODIUM IN THE VISCERAL MANIFESTATIONS OF ACUTE INFLAMMATORY RHEUMATISM.</b> By PROFESSOR JACCOUD . .	1
<b>DIGITALIS AS A DIURETIC.</b> By NESTOR TIBARD, M.D. (Lond.), F.R.C.P. . . . .	8
<b>OPIUM.</b> By J. S. TODD, M.D. . . . .	17

### Treatment.

<b>VARICOSE VEINS AND CONGESTED ULCERS; THEIR TREATMENT BY VENECTOMY AND MASSAGE.</b> By A. ERNEST MAYLARD, B.S., M.B. (Lond.) . . . . .	25
<b>CHICAGO SANITARY FLOUR FOR CERTAIN DYSPEPTICS AND DIABETICS.</b> By N. S. DAVIS, JR., A.M., M.D. . . . .	35
<b>THE TREATMENT OF WHOOPING-COUGH.</b> By PROFESSOR MARFAN, M.D. . . . .	38
<b>PLACENTA PRÆVIA; ITS DANGERS AND TREATMENT.</b> By J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S.E. . . . .	48
<b>A CASE OF TYPHOID FEVER COMPLICATED BY INTESTINAL HEMORRHAGE AND MENINGITIS DURING A RELAPSE, WITH REMARKS ON THE TREATMENT.</b> By HERMAN D. MARCUS, M.D., and JUDSON DALAND, M.D. (University of Pennsylvania), Philadelphia . . . . .	57
<b>CHLOROSIS: ITS COMPLICATIONS AND TREATMENT.</b> By CECIL Y. BISS, M.A., M.D. (Cantab.), F.R.C.P. . . . .	67
<b>THE TREATMENT OF CHLOROSIS.</b> By PROFESSOR HAYEM . . .	76
<b>THE TREATMENT OF PUERPERAL SEPSIS.</b> By WILLIAM H. WATHEN, M.D., LL.D. . . . .	83
<b>THE TREATMENT OF SCABIES.</b> By J. ABBOTT CANTRELL, M.D. .	91
<b>THE PREVENTIVE TREATMENT OF APPENDICITIS.</b> By ALBERT ROBIN, M.D. . . . .	98

**Medicine.**

	PAGE
<b>MYOCARDITIS.</b> By PROFESSOR E. VON LEYDEN . . . . .	105
<b>CLINICAL REMARKS ON A CASE OF ACUTE PERFORATIVE PERITONITIS DUE TO ULCERATION OF THE STOMACH.</b> By BYROM BRAMWELL, M.D., F.R.C.P. (Edin.), F.R.S. (Edin.) . . . .	116
<b>WEAK HEART; GASTRECTASIS FROM PYLORIC SPASM.</b> By CHARLES G. STOCKTON, M.D. . . . .	123
<b>CERTAIN POINTS IN THE DIAGNOSIS OF VARICELLA.</b> By ROBERT STEVENSON THOMSON, M.D., B.Sc. . . . .	129
<b>ENLARGEMENTS OF THE SPLEEN; FIBROID PHTHISIS; ENDOCARDITIS.</b> By NORMAN BRIDGE, M.D. . . . .	135
<b>ULCERATION OF THE PYLORUS AND ITS CONSEQUENCES; DILATATION OF THE STOMACH, WITH REMARKS AS TO THE TREATMENT.</b> By THOMAS OLIVER, M.A., M.D., F.R.C.P. . . . .	146
<b>ANEURISM OF THE ABDOMINAL AORTA.</b> By I. N. LOVE, M.D. . . . .	158
<b>AORTIC DISEASE OF THE HEART.</b> By GRAHAM STEELL, M.D. (Edin.), F.R.C.P. (Lond.) . . . . .	164
<b>PULMONARY TUBERCULOSIS; SPECIFIC PHARYNGITIS; AORTIC REGURGITATION.</b> By E. FLETCHER INGALS, M.D. . . . .	175
<b>TUBERCULAR PLEURISY; TACHYCARDIA.</b> By J. O. HIRSCH- FELDER, M.D. . . . .	180

**Neurology.**

<b>SPINAL IRRITATION.</b> By T. MCCALL ANDERSON, M.D. . . . .	198
<b>GENERAL PARALYSIS AS MET WITH IN HOSPITAL PRACTICE.</b> By ALEXANDER JAMES, M.D., F.R.C.P. (Edin.) . . . . .	209

**Surgery.**

<b>UMBILICAL HERNIA; REMOVAL OF A LEFT-SIDED GOITRE; CIRCULAR CRANIECTOMY FOR MICROCEPHALY; TU- BERCULAR ABSCESS OF THE HIP-JOINT.</b> By H. O. WALKER, M.D. . . . .	219
<b>EXTERNAL AND INTERNAL URETHROTOMY FOR RETEN- TION OF URINE; NEPHRORRHAPHY BY A FLAP METHOD FOR MOVABLE KIDNEY.</b> By J. D. THOMAS, M.D. . . . .	227
<b>RUPTURE OF A TUBAL ANEURISM OF THE FEMORAL AR- TERY.</b> By W. O. ROBERTS, M.D. . . . .	235
<b>SYPHILITIC STRICTURE OF THE RECTUM.</b> By JOSEPH M. MATHEWS, M.D. . . . .	239
<b>ACUTE OSTEOMYELITIS IN YOUNG CHILDREN.</b> By CHARLES GREENE CUMSTON, B.M.S., M.D. . . . .	249

**Gynæcology and Obstetrics.**

	PAGE
THE TREATMENT OF PLACENTA PRÆVIA. By A. PINARD, M.D. . . . .	264
EXCESSIVE MOBILITY OF THE UTERUS; PELVIC PERITONITIS AND FIBROID TUMOR; RETROFLEXION AND PROLAPSED OVARY; LACERATIONS AND CICATRICES OF VAGINA; AFTER-TREATMENT OF TRACHELORRHAPHY; RETROVERSION OF THE PREGNANT UTERUS. By PAUL F. MUNDÉ, M.D., LL.D. . . . .	276
EPITHELIOMA OF THE VULVA; RETROPOSITION OF THE UTERUS. By E. E. MONTGOMERY, M.D. . . . .	286
UTERINE HEMORRHAGE. By HENRY C. COE, M.D. . . . .	295

**Ophthalmology.**

THE RHEUMATIC AND GOUTY DIATHESSES AND THEIR RELATION TO DISEASES OF THE EYE. By WILLIAM OLIVER MOORE, M.D. (Columbia) . . . . .	303
OPERATION FOR THE EXTRACTION OF CATARACT AND FOR SECONDARY CATARACT; ASTHENOPIA FROM HYPERMETROPIA; PATHOLOGY OF SYMPATHETIC EYE-DISEASES; SYPHILITIC IRITIS. By THOMAS R. POOLEY, M.D. . . . .	318

**Laryngology and Rhinology.**

PARALYSIS OF THE MOTOR OCULI NERVE; ACUTE CATARRHAL INFLAMMATION OF THE EUSTACHIAN TUBE AND MIDDLE EAR. By SAMUEL G. DABNEY, M.D. . . . .	329
MASTOID CARIES WITHOUT EXTERNAL SIGNS; MASTOID SYMPTOMS FROM FURUNCLE; CARIES OF THE CANAL SIMULATING FURUNCLE AND TYPICAL MASTOID ABSCESS. By B. ALEXANDER RANDALL, M.D. . . . .	335

**Dermatology.**

A CASE OF EPITHELIOMA RECURRENS. By M. B. HUTCHINS, M.D. . . . .	340
--	-----



# LIST OF ILLUSTRATIONS TO VOLUME I.

## (EIGHTH SERIES.)

### PLATES.

	PAGE
Areas of percussion dulness of the heart, liver, and pleuritic exudation in a case of tubercular pleurisy (Fig. 1) . . . . .	182
A case of umbilical hernia (Fig. 1); the application of a continuous wire suture in the cure of umbilical hernia (Figs. 2 and 3) and the final closure of the wound with silkworm-gut sutures (Fig. 4) . . . . .	220
Ulceration of a large left-sided goitre (Fig. 5) . . . . .	222
Fibro-cystic goitre without evidence of malignancy (Fig. 6) . . . . .	222
The pathology of the recurrence of cancer (Fig. 1); horny epithelial growths into the lower eyelid (Fig. 2) and a mass of spindle-cells more highly magnified (Fig. 3) . . . . .	340
Pearly bodies in epithelioma (Fig. 4) . . . . .	342
Epithelial mass of round, oval, and spindle cells (Fig. 5) . . . . .	344
Characteristic appearance of an epithelioma (Fig. 6) . . . . .	344
Recurrent epithelioma (Fig. 7) . . . . .	346
Amorphous collection of epithelial cells in epithelioma (Fig. 8) . . . . .	348

### FIGURES.

Temperature charts of a case of typhoid fever complicated by intestinal hemorrhage . . . . .	64, 65
Typical pulse tracings in aortic stenosis (Figs. 1, 2, and 3) . . . . .	166
Two types of pulse observed repeatedly at different times in the same case (Figs. 4 and 5) . . . . .	166
Tracings from the right and left radial pulses of the same patient showing a marked difference (Figs. 6 and 7) . . . . .	167
A case of bisferiens pulse (Fig. 8) . . . . .	168
A case in which the obstruction and the incompetence seemed to neutralize one another (Fig. 9) . . . . .	168
A case of dilated aorta with slight incompetence of the valve (Fig. 10) . . . . .	168
A typical tracing of aortic incompetence (Fig. 11) . . . . .	168
Typical tracings of the pulse in aortic incompetence (Figs. 12, 13, and 14) . . . . .	170
Occasional loop-like tracing of the percussion wave in cases of regurgitation (Fig. 15) . . . . .	171
Bigeminal pulse from a case of aortic incompetence under the influence of digitalis (Fig. 16) . . . . .	171
Drawing of the left kidney in a case of nephrorrhaphy by a flap-method (Fig. 1) . . . . .	238





# Drugs and Remedial Agents.

---

## CONTRAINDICATIONS TO THE USE OF SALICYLATE OF SODIUM IN THE VISCERAL MANIFESTATIONS OF ACUTE INFLAMMATORY RHEUMATISM.

CLINICAL LECTURE DELIVERED AT THE PITIE HOSPITAL.

BY PROFESSOR JACCOUD,

Visiting Physician to Hospital Pitie, and Professor of Medicine in the Faculty of Medicine, Paris, France.

---

GENTLEMEN,—You may remember that in one of my preceding lectures I defined acute inflammatory rheumatism as a general disease, capable of localizing itself in a variety of ways. I told you at the same time that when this complaint takes a visceral form, salicylate of sodium must never be given. This remedy does not cure these manifestations of inflammatory rheumatism, nor does it even prevent them; and I shall now give you abundant proof in support of this statement.

Let us take, to begin with, the group of cerebral symptoms that constitute what is commonly called cerebral rheumatism. Experience has shown that these symptoms occur more frequently and more readily in patients who are being treated with salicylate of sodium. We have also found that if, when cerebral manifestations appear in a case of acute articular rheumatism, the salicylic medication is kept up the patient is practically certain to die.

I reported to the Académie de Médecine, in 1877, two cases of cerebral rheumatism that I had treated with salicylate of sodium at Lariboisière Hospital, in accordance with the enthusiasm that prevailed at that period for this remedy, and that both had a fatal ending. I showed, furthermore, that if the salicylate of sodium is discontinued the moment the cerebral symptoms appear, and cold baths are substituted in its place, this treatment is followed by recovery.

Since that date many other similar fatal cases have been reported in England, Germany, and elsewhere,—the greater number of such cases coming from England, as the most obdurate partisans of the salicylic treatment were to be found among the English practitioners. Consequently, make a careful note of the fact that you will be doing actual harm to any patient that you treat by salicylate of sodium if any cerebral manifestations of acute rheumatism are present.

Still, you must never lose sight of the fact that during the course of acute articular rheumatism the symptom delirium may occur, without its being in any sense a cerebral manifestation of the disorder under treatment. Thus, your patient may have the alcoholic habit, and the delirium may be of an alcoholic nature; or the patient may have been previously treated by sulphate of quinine, and the delirium may be due to the action of that remedial agent. It will not do, however, for you to wait until you are absolutely certain that your patient is showing signs of cerebral rheumatism before you interrupt the salicylic treatment; by so doing you will lose valuable time. As soon as you see any signs of delirium, stop your salicylate of sodium; to act in any other way will expose you to results that cannot be classed as catastrophes.

In the next place, let us consider another group of visceral manifestations of acute rheumatism, those that affect the cardio-pulmonary organs. In this connection, I also was the first to utter a word of warning by calling attention, at the same meeting of the Académie de Médecine, to the fact that, during the course of a case of acute articular rheumatism treated with salicylate of sodium, endocarditis or pericarditis can occur, and that they will be no more modified by the administration of this remedy than they were prevented by its use. Not many years elapsed from that date before medical opinion in almost all countries had sooner or later reached the conclusion that salicylate of sodium acts on the articular manifestations and on the rise of temperature in acute articular rheumatism; but that it has no action whatever on cardiac manifestations nor on cerebral rheumatism.

In visceral rheumatism, the evolution of the visceral disorder follows just the same course as though no salicylate whatever had been given; this remedy is, therefore, entirely without action in such cases. Since, on the other hand, it has a certain depressing effect

on the heart, its administration should be abandoned as soon as any cardiac symptoms manifest themselves. Out of twelve cases of acute rheumatism with cardiac complications that he treated with salicylate of sodium, Roussel lost two patients; and Church, who has also carefully studied this question, says that no treatment known has any effect on the tendency of acute articular rheumatism to localize itself on the endocardium and pericardium.

In every country these restrictions on the use of salicylate of sodium in acute rheumatism are now understood and accepted; but, in spite of this fact, many physicians still use this remedy in all such cases without distinction. The cardiac or cardio-pulmonary localizations of acute rheumatism do not admit of the use of salicylate of sodium, take my word for the fact, and if you continue to prescribe it, in spite of the appearance of such localizations, you will surely hasten the participation of the myocardium in the disease.

Since enthusiastic opinions are difficult to repress, the partisans of the administration of salicylate of sodium in each and every case of inflammatory rheumatism take refuge in the following statement. They say that they admit that the salicylate does not cure the cardio-pulmonary manifestations when these occur; but they claim that the use of this remedy, by moderating the intensity of the articular manifestations, and by shortening the duration of the complaint, prevents their occurrence.

Let us see what truth there may be in this statement. If the administration of salicylate of sodium could prevent the appearance of cardiac and cardio-pulmonary complications in acute rheumatism, the relative frequency of such complications ought to have diminished since this remedy was introduced into general practice. Nowhere are such statistics bearing on this point.

In 1881, Hood published the results of his experience. Among three hundred and fifty cases treated with salicylate of sodium, cardiac or cardio-pulmonary manifestations occurred two hundred and forty-four times, which gives a percentage of 68.85. Out of eight hundred and fifty cases treated by other methods, cardiac or pulmonary symptoms appeared five hundred times, a percentage of 58.82.

In 1882, G. Smith published the following results. Out of seventeen hundred and forty-eight cases treated with salicylate of sodium there were eleven hundred and nine cardiac or cardio-pul-

monary complications, a percentage of 63.4; seventeen hundred and twenty-seven cases treated by other methods gave nine hundred and forty-one such complications, or fifty-four per cent.

In 1883, Badt, out of one hundred and forty-eight cases where the salicylic treatment had been followed, reported seventeen per cent. of cardio-pulmonary localizations; whereas one hundred and seventy-six cases treated in other ways gave 16.4 per cent.

If now, we combine all these figures, we find that two thousand three hundred and thirty cases treated with salicylate of sodium gave fourteen hundred and forty cardio-pulmonary localizations, or 61.8 per cent.; while two thousand seven hundred and fifty-three cases treated in other ways gave fourteen hundred and sixty-eight instances of such complications, or 53.3 per cent.

The question has been, consequently, definitely decided: the use of salicylate of sodium in acute articular rheumatism does not prevent the occurrence of cardio-pulmonary localizations. If, therefore, you follow my advice, you will change your treatment as soon as you see the complaint assuming a visceral tendency.

Another aspect of the subject now presents itself. I explained to you on a former occasion why we are led to consider this disease as a general one, capable of diffusion, and why it is that the complications that arise—cardiac, renal, and others—are to be looked on as localizations of the disorder. The natural consequence of these views was to lead us to look into the bacteriological character that such a disease probably possessed, and medical opinion soon came to consider acute articular rheumatism as a microbiological complaint. No single micro-organism has been discovered that can be called specific of the disease; nevertheless, the bacteriological data we possess, owing to a certain degree of uniformity they show, throw sufficient light on this general character of the disorder, and I would therefore place acute articular rheumatism among the infectious diseases.

Babès found in the liquid taken from the knee-joint of a patient of this class micrococci and bacilli. Wilson, in 1886, found some also in the lung and myocardium. In the same year, Guttman detected the staphylococcus aureus in the knee, kidney, and pericardium of a rheumatic patient. Pétrone, in three cases of simple acute rheumatism, found micrococci which he looks on as similar to a microscopical element described by Klebs. In 1887, Mantle

again found an association of micrococci and bacilli in the articular exudation and in the blood; the micrococci frequently assumed the form of the diplococcus.

In 1885, 1886, 1887, we ourselves looked for micro-organisms in the articular liquid in acute articular rheumatism without complications, and failed to find any. This is not surprising, however, as the articular liquid is now pretty generally considered to be sterile in this complaint. In spite of this failure, I openly expressed my opinion, in my lectures at this clinic in 1887, that I looked on the disease as an infectious one that appeared to be connected in some way with the staphylococcus.

In chronic articular rheumatism other microbes are found. Researches made in Bern, in the case of a patient with chronic articular rheumatism, discovered in the endocardium, blood, and bronchial glands, a staphylococcus citreus, having all the properties of the common citreus, except that it was not pathogenic for the rabbit, guinea-pig, and dog.

Sallé, who also found a staphylococcus, raised the question as to why the joints suppurate so rarely in acute articular rheumatism. He was obliged to be satisfied with the explanation that since these agents, which are pyogenic under ordinary circumstances, have, in this complaint, their virulence lessened, their activity is not sufficient to produce pus. Other investigators have found the staphylococcus albus.

In short, we can say that in chronic articular rheumatism the liquid of the joints is sterile, but that the staphylococcus albus, aureus, and citreus can be detected in the tissues of the heart, kidney, and lungs.

Bousse reported a different microbe. He claimed to have found the pneumococcus, but he appears to have worked under special circumstances.

It is not without interest to note that in 1894 the author of a thesis on acute articular rheumatism found that by making an intravenous injection of a culture of staphylococci in animals he caused the joints to suppurate; whereas, when the experiment is made with microbes of weakened virulence, the ordinary symptoms of acute articular rheumatism occur,—that is, the production of serum only in the joints.

Of late years, since the conception of this disease as an infection

has been gaining strength, far greater attention has been paid than was formerly done to the conditions under which the cases originate. Thus we have perceived that in a large number of instances the disorder is preceded by some local process that might open the way for general microbial infection.

Among these various lesions the most important is a sore throat. Inflammation of the throat shows the same microbes as those noted in acute rheumatism,—staphylococci, micrococci, and sometimes pneumococci. The pharynx and tonsils may therefore be looked on as spots through which rheumatism frequently gains access to the system, in fact, it can be said that in a general way any acute, local condition can give rise to rheumatism, considered from this point of view.

I personally know of six cases in which the whole process was carefully followed. In one of them the microbe had penetrated the organism through a wound in the foot and in this case the staphylococcus albus was found; in the others the point of departure was a sore throat, complicated in one instance by purulent otitis.

This, gentlemen, is practically what we now know of the bacteriology of rheumatism. The researches to which I have alluded seem to show plainly that rheumatism is a microbial and consequently an infectious complaint, and that in articular rheumatism of the serous type the virulence of the microbes is diminished. It seems likely that variations in the degree of infection can explain the difference in symptoms that occur in clinical observation.

When I felt authorized, in December, 1887, in accepting the doctrine of the infectious nature of rheumatism, I was not able to base my opinion on bacteriological data, but was guided by the clinical evolution, by the capacity of diffusion shown by rheumatism. My opinion was also fortified by two cases—the only ones known—that show the possibility of the intrauterine transmission of rheumatism from mother to foetus.

The first was reported by Procope, in 1882, in the *Lancet*. The case was that of a woman, pregnant, and seized with acute articular rheumatism before labor set in; twelve hours after the child's birth its temperature rose, while the shoulder- and elbow-joints of one arm became red and swollen; the child manifesting its sensation of pain by continued crying. Recovery was obtained in a week by the use of salicylate of sodium.

In 1886, Sheffer published in a Berlin paper the case of a woman, thirty-five years of age, who, nine months pregnant, was seized on May 1 with a violent attack of articular rheumatism, her child being born five days later in a healthy condition. Three days afterwards the child's crying was so persistent as to require explanation, and the instep of one foot was found to be swollen. The following day one finger and a hip-joint were also found swollen. In spite of the administration of salicylate of sodium, this case did not get well for a long time,—the fever continuing until the end of May with the child, and until the middle of June with the mother.

The first symptoms appeared in the mother on May 1, and she was confined on the 5th; the infectious cause could therefore have only acted between the 1st and 5th. Since the first symptoms in the child occurred on the 8th, the writer thinks that the period of incubation must be from three to five days.

By considering all these facts, and the general course of an attack of rheumatism, you will probably be led to adopt my opinion, that this disorder is a general one, of the class of infectious complaints, and that it is very probable that the great clinical variations between different cases depend on equally great variations in the infectious agents.



## DIGITALIS AS A DIURETIC.

LECTURE DELIVERED AT KING'S COLLEGE HOSPITAL.

BY NESTOR TIRARD, M.D. (Lond.), F.R.C.P.,

Physician to the King's College Hospital and to the Evelina Hospital for Sick Children, London, England.

---

GENTLEMEN,—Many of the drugs used in the treatment of disease are employed almost as an act of faith. It is only of comparatively few that it can be said that they are used with any degree of certainty of the results which they are likely to produce. The large group under the former heading will include the remedies which are employed empirically,—remedies which are none the less potent, even though we are unable to furnish a satisfactory explanation of their precise mode of action. To the latter group, embracing drugs employed with comparative certainty, belong remedies which, according to an old term, are used rationally; they are marked by possessing certain definite actions, which may be estimated or measured both in the physiological laboratory and in the clinical wards, and on account of these actions they are applicable to diseases of which we know the exact pathology. Although such a classification is frequently useful, it must be admitted that the borderline is constantly changing, and that, if we follow the history of almost any drug which stands out prominently in the latter group, we shall find that in early days it was first employed empirically; in other words, clinical experience of the utility of a drug has often preceded any clear explanation of the precise mode of action.

Digitalis furnishes a very apt illustration of the above remarks. It has long been employed in medicine with a firm conviction of its potency and its utility; but, even at the beginning of the century, many of the statements concerning the drug differed in various textbooks, though perhaps the statements may, to some extent, appear incorrect in these days, owing to the different value attached to the terms employed: thus, for instance, for those who called it a cardiac

sedative, it is probable that attention was riveted almost entirely upon its power of reducing the rapidity of the action of the heart. It cannot, however, be doubted that the modes of employing this remedy were frequently much misunderstood, and that at the present time we are in a better position to decide upon the class of cases in which it is most likely to prove of service. This happy condition is mainly the result of clinical work and, secondarily only, of pharmacological research. Pharmacological research is always hampered by certain disadvantages. Foremost among these lies the difference of action on various animals: thus, it is well known that it causes the heart to stop in systole in cold-blooded animals and in diastole in warm-blooded animals. It has also been noted that it is very difficult to reduce the rapidity of the pulse in dogs by means of digitalis; and it has also been found that it possesses specially active powers in the dog, the rabbit, and the guinea-pig, while it possesses very little action in the snail and the toad.

The history of digitalis has also been complicated, owing to the difference of action produced in healthy and in diseased individuals: thus, even with regard to the diuretic influence of the drug, while there have been many who have maintained that it possesses little or no diuretic action in the healthy condition, its influence in increasing the amount of urinary flow in disease is beyond dispute; though in the history of the drug there has undoubtedly been some uncertainty as to the class of cases in which such diuretic action is produced. Moreover, it must be admitted that even in healthy human beings the drug does not always produce the same effects. In diseased conditions the action can be calculated with greater certainty, and it is to some extent dependent upon the different official preparations employed. Of the four active principles which are present in digitalis, one, digitonin, appears to be, to a large extent, antagonistic to the other three. Digitonin certainly causes some dilatation of the vessels, instead of contraction, and it is thought to be present in larger quantity in the infusion than in the tincture; to some extent, therefore, this will explain the preference which is often expressed for the infusion.

Dr. Lauder Brunton goes a step further, and suggests that the infusions vary considerably according to the character of the plants from which they have been prepared (*Action of Medicines*, 1897, p. 319). His reason for thinking this is that, in Edinburgh, the

infusion of digitalis is generally given in doses of half an ounce, while in London it is rarely employed in such large doses; and he thinks that the infusion, as employed here, must be made from plants possessing a larger proportion of active principles. Whether this variation of dosage really depends, as Dr. Brunton suggests, upon the difference in plants, or whether it may not be affected by racial distinctions and idiosyncrasies, forms perhaps too difficult a question to be considered here.

This diversity in different official preparations may or may not exist, but the action of digitalis is distinctly modified according to the dose employed, as well as according to the stage of the disease, and the effect is also, to a very large extent, dependent upon the duration of the employment of the drug. It is essentially these points, gentlemen, which I wish to impress on you to-day. I know that, in the examination-room, you would all probably be ready enough with answers to questions upon the action of digitalis, and presumably most of you would say that it acts in four stages, in accordance with the statements in one of the text-books, and that it essentially differs from strophanthus in the possession of diuretic properties. These statements you would no doubt have acquired in your first or second year, and, if you analyze them closely, you will see that they apply purely to physiological actions, and that they have no reference whatever to the action of the drug in disease. In all likelihood, only those who are working for the higher examinations would be prepared to make any detailed remarks on the action of digitalis as a diuretic, and upon the class of cases requiring diuretic action in which digitalis would be preferable to any other drug.

These questions are essentially questions of applied therapeutics, and upon the intelligence with which they are answered depends, to a large extent, the success of the medical practitioner. It is no use merely to be able to rattle through the four stages of the action of digitalis, or even to explain in which particular stage relaxation of the renal arterioles allows more blood to pass through the kidney and so produces diuretic effects. These statements, interesting in themselves, are of comparatively little importance at the bedside, where you see various results from the non-performance of the work of the kidney, and where you also see that we adopt very different measures to alleviate these different conditions.

I need hardly remind you of the long list of drugs classified as diuretics, nor of the various modes of favoring diuresis indirectly, without necessarily employing drugs which influence the kidney. You have probably noticed that the use of digitalis as a diuretic is not determined by a diminution in the secretion of water, nor even by the existence of dropsy, since you well know that in many cases of acute nephritis, especially scarlatinal nephritis, the secretion of urine is very greatly reduced at the same time that we have much œdema of the subcutaneous tissues of the body; and you know that, under these conditions, whatever other remedies we employ, we probably do not think of using digitalis. On the other hand, you will find that digitalis is frequently employed in advanced stages of kidney-disease, more particularly where there is some anasarca. In most of these cases the average daily amount of urine passed is somewhat reduced, sometimes even to a quarter or a sixth of the normal quantity; but here again I wish to warn you against the assumption that the use of digitalis as a diuretic in such cases is necessarily indicated by the diminution in the daily excretion of urine. Further, it may be necessary to remind you that digitalis is often employed where the dropsical conditions exist which are not necessarily dependent upon any organic renal changes.

What I want you more particularly to realize is that these apparently vague, hap-hazard employments of digitalis are not so much matters of chance as you might at first be tempted to imagine; and, moreover, that they depend largely upon the correct appreciation of the pharmacological actions of digitalis, and that they may be regarded as illustrations of the intimate relationship between pharmacology and therapeutics. Probably from the time that you left the class-room in which you first heard descriptions of the actions of digitalis upon the heart, one teacher after another has done his best to warn you against the employment of this remedy in every case of heart-disease; and has further indicated, from time to time, the grave results which may follow from the indiscriminate use of this drug. Perhaps you may have been tempted to wonder whether, after all, it was of any service in medicine, or whether it did not constitute a danger rather than a help. You are, no doubt, long past the stage when it was any temptation to employ this drug merely because a murmur had been recognized, and I hope that you have also realized that the question of the use of digitalis, so far as heart-disease is con-

cerned, depends not so much upon whether the mitral or the aortic valves are at fault, as upon the question whether the wall of the ventricle is performing its work efficiently; whether, in fact, the obstruction or regurgitation of either of these valves is sufficiently compensated by dilatation and hypertrophy of the walls of the heart, or whether the muscle is failing in its action and allowing of secondary engorgement. Even in the out-patient room one has often pointed out that, although either digitalis or strophanthus may be employed to reduce the frequency of the pulse and to restore its regularity, yet digitalis is used to a very great extent in cases of secondary engorgement of the lungs or other viscera, dependent upon the failure of the action of the ventricle.

This forms the clue to the use of digitalis as a diuretic. The conditions under which it may be appropriately employed are those in which dropsical effusions, or diminished secretion of urine follow from, or are associated with, failure of the work of the ventricle. Thus, to take the case of chronic kidney-disease which I have mentioned above, digitalis is serviceable, not in the comparatively early stages when the pulse tension is increased and when there is little dropsy, but in the later stages, when, as the general nutrition fails, the wall of the ventricle loses its power and becomes inefficient. Here we also find the explanation of the non-employment of digitalis in acute nephritis associated with general œdema, since, in these early stages of nephritis, there is generally very little fault to be found with the work of the ventricle; and, on the other hand, there is every reason to avoid the employment of a remedy which will increase renal engorgement. Although, in such cases, diuresis is desired, it is practically never procured directly by employing drugs to increase the amount of blood circulating through the kidney. You know full well that in such cases we use purgatives and diaphoretics, rather than diuretics, and that it is only when blood has disappeared from the urine that digitalis is likely to be used.

It is, perhaps, scarcely necessary to remind you that diuretics are required when the work of the kidney is indirectly interfered with as the result of disease in some other part of the body. Thus, for example, in cases of cirrhosis, or in cases with chronic pulmonary engorgement, as in emphysema and chronic bronchitis, or even in cases where, without valvular murmurs, the action of the heart is interfered with owing to weakness of its walls,—in any of these cases

the secretion of urine may be much reduced, and in nearly all the use of digitalis is often followed by beneficial results. Let me remind you that, in speaking of the four stages of the action of digitalis from the point of view of pharmacology, in the third stage the pulse increases in frequency, while there may be very little alteration, if any, in the work of the kidney; and it is in this third stage that I would enjoin caution in the use of the drug.

During the course of last autumn you may remember that there was a patient, H. S., aged sixty-one, in the Craven Ward, to which he had been transferred from the surgical wards. He was originally admitted on account of a bad carbuncle, and some time after this had been dealt with he developed symptoms of pneumonia, the temperature ranging between  $102^{\circ}$  and  $103^{\circ}$  F. At this time, that is, when the temperature was high, the urine contained a small quantity of albumen, a good many pus-corpuscles, and a few somewhat indefinite casts. From the onset of the pneumonic attack his condition caused much anxiety. The urine was passed involuntarily, and, therefore, could not be measured. There was frequent vomiting, the pulse was irregular, small, and unequal, and the breath-sounds were shallow, with crepitations scattered over the whole of the back. The patient was sleepless, and his mind appeared to be wandering. At the commencement, when the temperature was between  $102^{\circ}$  and  $103^{\circ}$ , the pulse ranged between 82 and 78, but, four days later, as the temperature fell, the pulse increased in rapidity to between 104 and 116. At first he was treated with small doses of strychnine, carbonate of ammonia, and the ether and ammonia mixture of the hospital.

In addition, owing to the weakness of the heart's action, eight ounces of brandy were given every twenty-four hours; but later, when the temperature fell to normal, this was reduced to four ounces. On the 7th of July, however, his pulse was found to be extremely irregular, and the temperature rose again to  $100^{\circ}$  F. Accordingly, the amount of brandy was once more increased to twelve ounces. During this time the urine was repeatedly examined, and we came to the conclusion that the amount of albumen was not more than could be accounted for by the pus which seemed to result from some cystitis owing to retention from an enlarged prostate. Although, under the influence of brandy, the sounds of the heart became more clear, the pulse-rate rapidly increased from

between 80 and 100 to an average of 130 to 140; and at one time, when the rapidity was counted with the stethoscope, it amounted to 170. With this rapidity of pulse there was some slight increase of pneumonic signs and crepitation over the lung, with faintly tubular breathing at the right base. There was also some crepitus and dullness at the left base. These signs gradually cleared up, and the temperature fell. The pulse, however, did not regain the normal rate, nor did it correspond with the sounds of the heart. Thus, on the 4th of August we found that the contractions of the ventricle averaged from 150 to 160 in the minute, while the pulsations which reached the wrist were only 100. Up to this time there had been a gradual diminution in the daily amount of urine passed. During the pneumonic attack it had averaged from sixty to seventy ounces, and by degrees fell to from thirty to forty.

In view of the rapidity of the heart's action, and of the small amount of urine being passed, I prescribed fifteen minims of the tincture of digitalis on August 6, and in doing so gave the warning that this amount should be reduced if it was found that the pulse became more rapid or that the daily excretion of urine diminished, and I indicated that probably both of these changes would occur. At first the pulse fell to 88, while the amount of urine increased from thirty-two ounces to sixty-four on August 8; but on the 12th and 13th it was found that the pulse had once more increased to from 96 to 100, while the amount of urine had fallen to thirty-three and thirty-eight ounces respectively. On the 13th, therefore, the dose of digitalis was reduced to ten minims, and under this dose the heart's action remained fairly constant at from 88 to 96. The amount of urine, however, was still considerably below the normal quantity,—namely, from thirty to forty ounces,—and accordingly, on August 20, diuretin was given in twenty-grain doses three times a day. On the 21st the amount of urine had risen to sixty ounces, but it fell to forty-four on the 22d, to thirty ounces on the 23d, and during the next six days it ranged between thirty and eighteen ounces. On the 29th we once more resumed the administration of digitalis in ten-minim doses, and I combined with this five minims of liquor strychninæ. Under this treatment the heart gradually recovered power, and the urine, when it could be measured, was passed in more satisfactory amount. The patient was so well, how-

ever, at this time that he was able to be up and walk about the ward without distress.

It is somewhat interesting to compare these results with those noted by Dr. C. R. Marshall (*Journal of Physiology*, vol. xxii., September 1, 1897), who found, as the result of experiments, that while digitalis tends to diminish rather than to increase urinary secretion, nitroglycerin acts as a diuretic after the blood-pressure has returned to the normal, and that a combination of both substances is probably a more powerful diuretic than nitroglycerin alone. He found, by experiments upon rabbits, that during the time that the blood-pressure is reduced by nitroglycerin there is a notable fall in the amount of urine; but that if digitalis had previously been administered, causing the fall to be of a more transient character, scarcely any diminution in the urine occurred.

In the paper above referred to Dr. Marshall gives a valuable summary of the differences of opinion connected with the diuretic action of digitalis, and he gives reasons for believing that when this drug causes a diminution in the urinary excretion in dogs and also in rabbits, this diminution is probably the result not only of an irritant action upon the renal tissue, but also of a diminution in the amount of blood passing through the kidney owing to contraction of the arteries. He maintains that when, in disease, a diuretic effect is obtained with digitalis, it is almost exclusively confined to cases of heart-failure, and he thinks that in these cases the increased urinary secretion is probably wholly dependent upon the circulatory changes, and very slightly, if at all, upon true renal stimulation.

These suggestions are wholly in accord with clinical experience of the use of digitalis, and you will have realized, from what I have already said, that the true place of digitalis as a diuretic is to be determined by the recognition of cardiac weakness, rather than upon a diseased condition of the kidney. Hence, in the selection of a diuretic to be employed in any particular case, it is essential that the condition of the cardio-vascular system should be considered before digitalis is used, and that this drug should only be given if there is fair ground for the assumption that cardiac weakness, as shown by the nature of the pulse, or by the rapidity of the heart's action, is playing an important part in the production of the main symptoms. You will now understand why digitalis should not be used in the early stages of acute nephritis, where any increase in the



force of the contractions of the heart will be likely to increase renal engorgement. You will also recognize and appreciate the reason for avoiding the routine employment of digitalis in ordinary cases of chronic nephritis so long as there is increased blood-pressure within the arterial system, since, under such conditions, by further increasing the blood-pressure digitalis will be likely to do harm rather than good. Moreover, I may remark here that the increase in the blood-pressure, under such conditions, might favor the onset of hemiplegia, through the rupture of an atheromatous vessel. Finally, it is not enough merely to recognize the diminution of the daily excretion of urine or irregularity of the heart's action as reasons for the administration of digitalis. The drug should only be given if these two conditions can be assumed to have some definite causal relationship.

## OPIUM.

RECOLLECTIONS FROM A SERIES OF FOUR LECTURES ON OPIUM DELIVERED AT  
THE ATLANTA MEDICAL COLLEGE.

BY J. S. TODD, M.D.,

Professor of Therapeutics in the Atlanta Medical College; First Assistant Surgeon-General of the United Confederate Veterans, etc.

---

GENTLEMEN,—You will observe that I have written upon the board that which you students facetiously term my text.

Opium relieves pain, agitation, delirium, and spasm, thereby making sleep possible; it restrains tissue change, lessens all the secretions except those of the skin, represses hemorrhage, and both directly and indirectly sustains the powers of life. These are almost the exact words of the learned Stillé.

Would that I knew more of the early history of this drug, which, possessing these manifold virtues, is crowned as the chief of therapeutic agents, and from a medical stand-point the magnum donum Dei.

If used by the ancients at all, it was under another name, and from the virtues ascribed by Hippocrates to some of the mydriatics as pain-relieving agents, I am often constrained to think that his lactucarium was opium.

Morphine was the first alkaloid separated from a crude drug, and was discovered by Sertürner, a German chemist, in 1816.

Sydenham, when he found he could extract the virtues of opium with alcohol, exclaimed, "Laudus Dei!"—laudanum!

Within its sleepy depths not only resides morphine, the prince of pain, but this latter drug has been decomposed, and we have apomorphine, the most immediate of all emetics; and while opium is the chief of antispasmodics, thebaine, a convulsant equal to strychnine, is also a constituent.

Therefore, in what I have to say to you, when I say opium I mean opium, and not its derivatives. Bear in mind that morphine

kept in aqueous solution for any length of time is apt to be decomposed and converted into apomorphine, which accounts frequently for the vomiting that occurs when the former is administered.

Pain is not an unmixed evil, and while it is the highest and most grateful prerogative of the physician to relieve suffering, it should not be done generally until the cause is found out. For instance, to quiet a pain in the belly, when a symptom only of a strangulated hernia or an incipient appendicitis, would be in some instances a fatal mistake. But pain by itself, uncombated, deprives the patient of sleep, and the long list of evils that ensue are competent to produce death.

The pain of an extensive burn, the pains of labor, pains which children bear so badly, not only deprive the patient of sleep, but often produce convulsions, which may end fatally. Pain is the first herald of inflammation, for where there is an irritation there is a determination of blood to the part, which goes on into congestion, etc. Relieve this irritation at its beginning, and the determination ceases; and often the congestion and consequent inflammation does not ensue, and you have jugulated the disease at its inception.

Sleep is impossible when we are in pain; many physiological changes cannot and do not occur unless we are asleep. Sleep is not rest simply, not absence of voluntary motion only, but it is more than this. To strap a person supinely upon his back and render him immovable would not put him in the condition called sleep. "Dreamful sleep is a ligation of the senses and a liberty of reason." During sleep, said an old Roman philosopher, the soul is dismembered from the body, and seeks a dusky workshop far away, where it receives repairs necessary to sustain the body in the waking hours. Good sleepers are long lived. "Balmy sleep is nature's sweet restorer," and it is seldom good policy to awaken your patient from refreshing slumber to administer medicines, and more especially if that patient be a child.

Dr. Davidson,<sup>1</sup> of this State, in 1885, first announced the antagonism between morphine and nux vomica. While I do not believe that strychnine will supplant atropine as an antidote to poisoning by the drug we are discussing, yet I am sure that the antagonism existing between the two renders it an exceedingly valuable dis-

---

<sup>1</sup> Atlanta Med. and Surg. Journ., December, 1885. My attention was first called to strychnine in abdominal surgery by Dr. L. D. Johnson, of Whittier, California.

covery, and robs morphine of the dangers after operations in abdominal surgery that attends its use when given alone.

When a laparotomy is performed, especially for the ablation of the ovaries, the shock which follows is often not only dangerous, but the pain is heart-rending, excruciating. Given such a case, I have never seen, though my experience has been limited, ill effect follow if strychnine is injected, with just sufficient morphine to relieve pain. The paralysis of peristalsis, which the surgeon tells you ensues from morphine, was obviated when strychnine was given with it in every case which has come under my observation where a laparotomy has been necessary. The danger of paralyzing the heart by overstimulation with strychnine, even in large dose, is obviated when combined with morphine.

Strychnine, being equally as good a heart stimulant and as lasting in its effects as atropine, not only prevents the feeling of depression which morphine when dying out occasions, but is preferable, when atropine is used with the latter, where dryness of the mouth, fauces, or skin is a sequelæ.

I am happy to state that, in conversation with Dr. F. W. McRae, a former student, now professor of surgery in the Southern Medical College, he tells me that in nineteen consecutive successful operations for appendicitis he has used a sufficiency of morphine to relieve pain, conjoining it with strychnine, and always with the happiest results.

Again, suppression of urine has not been a symptom in any of our cases.

Strychnine is a synergist of morphine in its action on the heart, and overcomes, without interfering with its pain-relieving power, the obtunding of the reflexes caused by the latter drug.

Strychnine is one of the most important remedies, as a substitute for opium or morphine, when we have that unfortunate habit to treat, it being a stomachic and nerve tonic, appetizer, etc. Strychnine determines blood to the nervous centres and heightens the reflexes; morphine the opposite. Morphine contracts the pupil. Strychnine enlarges the visual field and sharpens the acuteness of vision. Morphine causes diaphoresis, but strychnine is an anhydrotic. Morphine depresses not only the sexual appetite, but the bodily functions, mental and corporeal generally. Strychnine has the opposite effect.

A narcosis so profound that it would in the large majority of cases be inevitably fatal if the patient had taken morphine, would be recovered from if opium had been administered instead. Why? If you will watch the manner of recovery from an excessive dose of opium, you will be given the key: notice the convulsive movements,—sometimes a general convulsion; they are caused by the thebaine, which is the powerful analogue of strychnine, and is a constituent of the crude drug.

It is an old adage, that is true in the main,—viz., in the essential fevers opium should not be used at their beginning, or at their acme, but rather during the augment to appease their violence, and during the decline to soothe, sustain, and strengthen the patient. For in the beginning of these affections there is paralysis of secretion, and some such agent as mercury, which stimulates all the secretory and excretory functions of the body, should be given to assist nature in expelling the *materies morbi*. If we only notice, then, that the skin is dry, and give opium because it is a diaphoretic, we forget that the constipated bowels, the lessened liver, kidney, pancreatic, bronchial, nasal, and internal secretions all say nay to the proposition. It restrains tissue change and sustains all the powers of life; therefore, from a therapeutic stand-point, it is of inestimable value in all self-limited diseases. Not a chemical food, not containing the elements capable of being metamorphosed into tissue, but, by causing us to live more slowly, it preserves the tissue on which the vitality depends until the disease has run its race. A man who could live on water alone for twenty days, we surmise by the restraining influences that would be exerted if opium had been administered with the water, would live so much less rapidly that life would be prolonged for twenty-five days. And in diseases of this character it is doubly true "that every moment of time is a moment of mercy." In all self-limited diseases there is a contest between the strength of the patient and the life-history of the disease, so we have been taught in the past. But this statement, I think, is subject to qualification, in view of the physiological functions that are being rapidly discovered of the ductless glands. I predict that the future will demonstrate that opium does not interfere with the secretion from the ductless glands, those organs that supply the neuclein, which destroys the specific cause of the disease, and whose elaboration, emptying directly into the blood-current, is the explanation of the so-called self-limitation of so many affections.

As regards the antagonism existing between many medicines and the toxins generated in the body, little is known, and the field for investigation is vast, for whether the ptomaines or germs are the hurtful factors is still sub judice. But we know—common-sense teaches—no germ, no ptomaine!

The hibernating animals are not only at rest, but profoundly asleep (the reptiliæ) during the winter months; hence the adipose tissue with which they begin their long fast is sufficient to keep alive the vital spark, and they come forth lean, it is true, but responsive to the voices of spring. Cold narcotizes them more completely than morphine does man.

That morphine so obtunds the nervous centres to the toxic influence of urea that its retention does not produce convulsion is so manifest that, under the teaching of the great Loomis, it has become the orthodox remedy for that symptom of the non-elimination of this excretory product. And here, in this connection, I wish to remark that in the treatment of poisoning by milk (tyro-toxicon, the ptomaine found by Vaughan), producing cholera morbus, with cramps, collapse, etc., of such serious nature that if you do not administer morphine, in the large majority of cases—at least such is my firm conviction—many of the patients would die. Now, I know that among the most scientific it is argued that the vomiting and purging are but efforts of nature to get rid of the cause, but so violent are the symptoms and agonizing the pain that, unless you relieve them with morphine hypodermically, fatal results will accrue; if you stand by idly, the disease will eliminate the patient.

In the treatment of cholera in its early stages all agree that in the premonitory diarrhœa opium and bismuth constitute the best treatment, but after the disease is pronounced there is want of harmony as to what is the best drug to administer, so that various and widely diverse remedies have been resorted to, from croton oil to astringents. But the consensus of medical opinion is that hypodermic injections of morphine—and from a theoretical stand-point I would give strychnine and morphine, because there is a paralysis of the vasomotor nerves—constitute the very best treatment, for we know, in this disease, that nature is incapable, in the majority of cases, of eliminating the poison.

Dysentery and diarrhœa, especially in Southern climates, are among the most common and fatal, therefore the most interesting, diseases that you will have to treat. To give you an idea of the

great prevalence of the disease in the Union army during the four years of fratricidal strife, one hundred and one thousand Federal soldiers were killed on the battle-field; ninety-six thousand died from affections of the alimentary canal; I may safely say ninety thousand from dysentery and diarrhœa. Only eleven thousand more were killed than succumbed to these two affections! Now, why do I mention this? It is to impress upon you that while opium is a *sine qua non* in the treatment of these two affections, after the cause has been removed, yet without the proper diet it is totally inadequate. The Federal surgeons had opium in abundance, but milk could not be obtained. Now let me imprint indelibly upon you (and illustrate it with a case) the importance of remembering that giving a drug is not the end of the matter; for a medicine may be *in a man, but not of him*. Neither opium nor morphine are astringents locally applied, though they are the most powerful of this class when absorbed; but they must enter the circulation in order to produce their full medicinal effect.

A gentleman applied to me with dysentery; he had been purged freely, but was still suffering from the characteristic griping and small mucous actions, tinged with blood. I prescribed morphine three grains, water three ounces, and directed that he take one teaspoonful after each action. In twelve hours he sent for me and said he had spent a most miserable night, being frequently disturbed by the action of his bowels. I asked him how many motions he had had? He replied by asking me how many doses of medicine there were? I told him twenty-four. He said, "Well, I have just taken the last dose!" I then injected hypodermically one-eighth grain of morphine; it was six hours before it was necessary to repeat it.

I witnessed my first post-mortem over thirty years ago. The case was one where the subject had died of dysentery. Among other things revealed by the autopsy were thirty one-grain opium pills in the alimentary canal. Tolerance to drugs—so-called—in a number of diseases is often their non-absorption.

In chronic diarrhœa, or dysentery, the milk diet, Rock Bridge alum water, and small doses of opium given cautiously,—very cautiously, lest your patient acquire the opium habit,—has proved so invariably successful with me that I have come to treat these cases with a confidence born of success, unless the affection is caused by *amœba*, consumption, or cancer.

In the treatment of enteritis, morphine *hypodermically* injected, and predigested milk or buttermilk,—for sweet-milk is wholly digested in the small intestines,—offer the best hope of cure and constitute the main treatment.

In hemorrhages, especially of the bowels and lungs, I do not see how I could get on without morphine,—quieting the cough on the one hand, and stopping the peristalsis on the other, thereby fixing the clots in the mouths of the bleeding vessels, and also calming the nervous agitation of the patient, for the sight of blood always inspires terror.

In the treatment of pneumonitis—not pneumonia, but traumatic inflammation of the lungs—opium has been so uniformly and invariably successful with me that I could not conscientiously use or sanction the use of any other medicament. If I am correctly informed, this treatment for wounds of the lungs was an accident; for, up to the battle of Gettysburg, wounds of this structure were regarded as almost certainly fatal. There were so many wounded to be cared for after this sanguinary conflict that those wounded in the lungs were simply given opium in sufficient quantity to let them die without pain, and those hurt in other parts of the body, where there was a hope of saving their lives, were attended to. To the astonishment of the surgeons, the large majority of all those wounded in the lungs, so treated, recovered.

You remember, at the battle of Crécy, the exhaustion of the supply of boiling oil as a treatment for gun-shot wounds first demonstrated that it was unnecessary.

In the treatment of abortion the hypodermic injection of morphine, conjoined with rest in the recumbent posture, has prevented the mishap, in my experience, so often that I have ceased to consider that an abortion is inevitable until I have tried it persistently.

Opium for a long time was regarded as the best and most rational treatment for peritonitis; so thought I, under the teachings of the old masters, and but a few years ago in my lectures advised its use unconditionally. But when clinical fact is at variance with theoretical deduction, you must always abandon the latter, however much you are wedded to it. The salines are the best single treatment, but are not specifics; for when the reaction comes about—after the fashion of giving salts in every case subsides, when the statistics which time will furnish are at hand—there will be differ-



entiated for us by the clinician and pathologist certain cases having distinctive features that we will successfully treat with opium, as of yore, that purgatives are fatal in.

There is a form of dyspepsia, not at all uncommon, characterized by considerable pain in the stomach soon after eating, sometimes vomiting, and generally diarrhœa, that calls for opium. This irritable condition of the gastric mucous membrane can be soothed best by giving a hard opium pill, one-fifth to one-third of a grain, say fifteen minutes before eating. I have often relieved this kind of indigestion by this procedure after failure with other approved methods. The impairment of the appetite occasioned by the opium is salutary, as persons so suffering eat too much.

In the distressing dyspnœa accompanying grave cardiac affections, despite the presence of albumen and casts in the urine, you should not hesitate to inject morphine and atropine, for, as Dr. Loomis long ago remarked, it gives the power to breathe. After the failure of digitalis and all other diuretic synergists to arouse the kidneys,—they very probably often causing spasm in the blood-vessels and nervous exhaustion from over-stimulation of this emunctory,—morphine and atropine injected will sometimes restore their function. So again, by relieving spasm of the circular fibres in the intestines, the same proceeding, while not actually acting as a purgative, permits catharsis by restoring the patulous condition of that canal.

The inco-ordinate pains of labor are best treated in the same way, and when there is also, as is generally the condition, a rigid os uteri, do not forget to give by the mouth every fifteen minutes until decided nausea is produced one-fourth grain doses of a now nearly obsolete remedy,—tartarized antimony.

Finally, gentlemen, all the living must die, and to “shuffle off” this mortal coil calmly, peaceably, as if falling asleep, is much to be desired; therefore, when the cold dews of death wet our brows, when our feet are already in the icy Styx, when the pale messenger comes and demands his victim, we can still cheat the King of Terrors, for,

“ When, O Death ! thou would'st carouse,  
Bind the poppy round thy brows ;  
Bind it round with bonny glee,  
'Tis the fittest wreath, O Death ! for thee.”

# Treatment.

---

## VARICOSE VEINS AND CONGESTED ULCERS: THEIR TREATMENT BY VENECTOMY AND MASSAGE.

CLINICAL LECTURE DELIVERED AT THE VICTORIA INFIRMARY, GLASGOW.

BY A. ERNEST MAYLARD, B.S., M.B. (Lond.),  
Surgeon to the Victoria Infirmary, Glasgow, Scotland.

---

GENTLEMEN,—The subject of our lecture to-day is a disease the simplicity and frequency of which may tempt you to regard it as worthy of little interest; and yet among the so-called innocent complaints there are few more instructive in their etiology and pathology, more tedious and troublesome in their natural course, and yet withal more amenable to rational treatment. If the number of cases which you have seen in the wards do not impress you, your own experience in private practice soon will. And you will there find that to successfully relieve a sufferer of an old standing complaint, which is and has been a constant source of worry and pain, is both the means of conferring a blessing and receiving one. So, kindly bear with me while I strive to describe to you, first, the interesting features connected with the etiology and pathology of the disease before discussing the subject of treatment, for it is only by a correct appreciation of these aspects of the complaint that we can supply what I have designedly expressed as its rational treatment.

I shall not take up your time by describing the purely clinical aspects of varicose veins of the lower extremity; you are sufficiently familiar with them; you know that these vessels are mostly tortuous, dilated veins, either normal or adventitious, connected either with the long or short (internal or external) saphenous veins. The valves which normally exist in their channels are no longer capable of supporting the column of blood above, hence gravity becomes a cogent factor in producing undue distention. But we must ask ourselves, in

a preliminary investigation of the subject, What are the causes which primarily lead to these abnormal dilatations? For no treatment can be called truly rational that does not deal with and fully consider the origin of the disease it proposes to remedy.

#### ETIOLOGY OF VARICOSE VEINS.

We may safely ascribe the causes of varicose veins to either congenital or non-congenital influences. There is but little doubt that the large majority of those cases which we meet with in the young and adolescent are congenital; and the same explanation applies also to many of those met with in adult life. What may be the causes, pathological or developmental, at work during intrauterine life does not concern us here; we have simply to deal in this particular class of cases with results, the causes of which, having passed, are without our power to prevent or to remove. Not so, however, is it with those cases which we have classified as the result of non-congenital influences. In these there is some definite cause to which we can ascribe the dilatation and tortuosity of the vessels. If we say that this abnormal condition is dependent upon some obstruction to the return flow of blood from the extremity, our attention is first directed to the anatomical course taken by the affected vessels, and, secondly, to the agents which may press upon them or interfere in some way with the unimpeded flow of blood through them. The course taken by the internal and external saphenous veins is, as you know, a perfectly superficial one. Embedded in the subcutaneous cellular tissue, they have nothing but the cutaneous structures to cover them. Hence these veins are specially predisposed to external pressure. Man, in his endeavor to adapt a costume most suited to his convenience, has strangely erred in not always selecting one best considered for his welfare. The tightly gartered stocking, or the closely buckled knickerbocker, are both means of causing harmful constriction. It is not unworthy of note in this respect that woman, in—shall I call it—her newly emancipated position, while striving after greater liberty in the matter of clothes, is, under what she calls a rational costume, committing an error liable to lead to greater ills than those from which she is seeking to free herself. The ostensible reason of this “improved” method of dressing is the greater freedom of movement which it allows; but it is this increased activity which so very materially augments the harmful effects of pressure upon the superficial veins. Women at all times are those most prone to vari-

cosity of the veins, so that we may confidently expect a considerable increase in this class of patients, unless the promoters of this so-called rationalistic movement can see their way to devise means of removing all constricting agents from the surfaces of the lower extremities.

Irrespective, however, of direct pressure applied to these veins as they course along the limb, we must consider the possibility of obstruction to the upward flow of blood in any region of the vein trunks. You will remember that the femoral veins become continuous with the external and common iliac, hence any pressure upon these vessels will produce precisely similar results to those connected with what we have considered in the case of the saphenous veins. You will have no difficulty in conjecturing what is likely to be the commonest source of pressure upon these trunks. The anatomical relations of a loaded rectum or a gravid uterus will at once strike you as causes, and as additional reasons for the greater frequency of varicose veins in women than in men.

#### PATHOLOGICAL SEQUELS TO VARICOSE VEINS.

But we must now leave the subject of causation, to consider the pathological consequences of varicose veins. It would lead me too far away from the end I have in view if I were to discuss fully each of these. Briefly, they are, œdema of the foot and leg, eczema, hemorrhage, heat, irritation, pain, and ulceration. It is the last to which I wish particularly to direct your attention.

The healthy condition of any part of the body, be it skin or deeper structures, depends upon the free circulation of normal blood through it. To, in any way, interfere with that freedom of circulation is to impair the vitality of the part. Hence, sooner or later, according to the degree of impairment, the tissues comprising the affected part must degenerate and eventually break down. It needs only some additional exciting cause, such as traumatism in some shape or form, to initiate the process, and when once started the lesion extends until an area is reached where the tissues are of a more healthy character.

Now, the particular lesion I here refer to is ulceration; in other words, we obtain, as a result of the impaired vitality, what is known as a varicose, chronic, or indolent ulcer, or, as I have preferred to call it, a congested ulcer. Let us for a few minutes examine a little more closely the pathological processes which lead to the formation

of this ulcer, for it is only by properly understanding these that we are guided towards the adoption of a rational treatment.

#### PATHOLOGY OF CONGESTED ULCER.

The result of obstruction to the return of the venous blood from the skin and subcutaneous tissues is the engorgement of the part both with stagnant blood and with exudates. Hence there is, in addition to the hinderance to the entrance of arterial blood, a check to the removal of used-up and effete material by the veins and lymphatics. Such a stagnation of the normal functions of the part as results soon leads to a break-down. For unless nourishment is freely conveyed to a part, and the waste products as freely removed from it, we cannot expect that life can be maintained any more in the case of the skin than with the heart, lungs, or any other organs or structure.

It is sufficiently familiar to you that ulceration in connection with varicose veins is situated at some portion of the circumference of the leg above the ankle. Most frequently we find the ulcer located on the inner side. The reason for this seat seems at first sight somewhat puzzling. We should naturally expect that, the foot being the most dependent part, the changes which we have been discussing would have been most marked there. With the exception, however, of cedema, such is not the case. We never meet with ulceration below the ankle. The explanation of this *lieu d'élection* is thus given by John Hilton in his well-known work on "Rest and Pain": "The superficial and deep veins of the leg freely communicate with each other in the neighborhood of the ankle-joint. The first two inches above that point is the spot where the greatest stress is laid upon these superficial veins; below that point they freely communicate, and if the blood cannot return by the superficial veins, it can do so by the deep veins or *vice versa*. But when you reach the point where that brown patch of skin so often occurs in old persons, above the inner malleolus, the anastomoses are less free, and this appears to me to be the reason why ulcers from varicose veins occur so frequently about that neighborhood."

To summarize briefly, then, what has been stated: Non-congenital varicose veins depend upon some obstruction to the upward flow of blood, and these veins, whether congenital or non-congenital, are the cause of ulceration. All treatment, therefore, resolves itself into an attempt at removal of these respective causes,—in the one

case, of any source of obstruction; in the other, of the veins themselves when possible.

#### TREATMENT BY VENECTOMY.

I need not dilate further upon the obstructive causes, but ask your attention regarding the treatment of the veins. It is not every case that lends itself to the radical measure of excision; while in one case you may find only one large and long dilated tortuous vessel, in another you may have diffuse congeries of more or less distended vessels; between these two extremes you will meet with every degree of varicosity, involving only one vessel or several.

If, then, your case is one where operation is not possible, or, if possible, only to so very limited an extent that what is removed will not sufficiently relieve the congested area, you must adopt some conservative measure. Nothing, in this respect, acts more efficiently, and with better results, than the careful and judicious application of pressure by means of a Martin's rubber bandage. I will apply the bandage to this patient's leg, in order that you may see how it is done. You will observe I commence just at the base of the toes, and then continue by the circular method to cover the whole foot and leg as far as the knee, beyond which, in this case, the varicose veins do not extend. I place just sufficient tension on the bandage to make it set evenly on the surface and each turn slightly overlaps the other; no reverses are used. My object is not so much to exert pressure as to give support. The patient very easily learns to do this for himself or herself, and is directed to take it off at night, but always to wear it when up.

Now, in the next case which I show you, and upon which I intend to operate, you will observe we have some three or four large varicose veins extending up one leg as high as the saphenous opening. The most marked varicosity is observed on the inner sides of the knees in both legs. This is in every way a suitable case to operate upon, and the way I shall proceed is as follows: I shall make incisions six to eight inches long, and possibly longer, over the most prominent parts of the veins. The course of the incisions will be along that of the vessels. In the case of the vein which is varicose as far as the saphenous opening, one end of the incision will be opposite that opening. If there is no thickening or œdema of the skin over the vein, I shall expect to be able to slip the director easily along the superficial fascia immediately over it, and then, dividing this with a

knife, at once expose the vein. I shall next separate the exposed portion at its distal extremity, and ligate it with a silk ligature applied at two points, so that the vein can be divided between. The proximal end will be then treated in the same way. The intermediate portion, being thus freed at its two extremities, will be dissected up, and all veins entering the main trunk clamped or ligated as deemed advisable. The skin edges will be finally stitched together, and, if all goes well, the dressings will not be removed for a week, when the stitches will probably be taken out. This same operation will be performed at two or three other places on the limbs. As illustrating somewhat the result we may hope to get, I will briefly narrate the case of a young man whom I operated upon about fourteen years ago, who had the most extensive varicosity of the veins of both legs I have ever seen. Enormously distended veins extended from the ankles to the groins. He was much hampered in his movements, and unable to take much exercise. I removed three long pieces of varicose vein from each limb, a segment in both cases being taken from close to the saphenous opening. About a year afterwards I had a letter from him, stating that he was now able to swim, a pleasure he had never hitherto been able to indulge in, although residing all his life at the coast. I mention this as a somewhat extreme case, but it illustrates equally the good results you may hope to get in all properly selected cases.

I have no intention of describing other operative measures, but I want to say a few words upon the advisability of removing long pieces of veins. If you remove a comparatively long piece, you are sure to ligature several venous radicles, which would otherwise remain intact and prove a continuous source of engorgement to the very part you are seeking to relieve. The operation, if performed with strict antiseptic precautions, is not rendered one iota more dangerous than in the case of more limited excisions, nor does it add materially to the length of time of healing. It is incomparably a better operation than either injection, subcutaneous ligature, or the removal of small segments in several places.

While I have described to you the method of removing large varicose veins in uncomplicated cases, there are other instances where excision is equally advisable, although the operation will be much more difficult to execute. Thus you may have to dissect out localized plexuses of veins, or you may have the skin thickened,

inflamed, and adherent to the veins, so that it is all but impossible to free them without puncture. In these latter cases it is better to employ a tourniquet, although the use of this renders it somewhat more difficult to distinguish the veins from the surrounding tissues.

#### TREATMENT OF CONGESTED ULCERS BY MASSAGE.

We must now leave the subject of varicose veins to discuss that of congested ulcers. You will doubtless clearly have concluded, from what I have stated regarding the pathology of these ulcers, that all they need is to be relieved of their congested condition. They are typically "sluggish," and want stimulating, not, as erroneously supposed, by some external application, but by every measure that will tend to restore a normal circulation through the part. The only stimulation that is needed is that which would be supplied by the free entrance of arterial blood. But this can only be effected by removing those conditions which we now know to be the source of obstruction.

In the first place, then, we consider and treat as far as possible any hinderance to the upward flow of blood through the main venous channels.

In the second, we place our patient in the recumbent position, in order to remove the effect of gravitation upon the abnormally superimposed column of blood.

In the third, we excise the veins as above described, if the case is suitable for operation.

In the fourth, we treat the ulcer itself. And here let me first show you a simple method of recording the shape and size of the ulcer, and the progress made from time to time in its treatment: Take a piece of clean white or pink blotting-paper, a little larger in size than the ulcer, and press it evenly over the part. On its removal you will have a more or less perfect outline of the ulcer. Cut round this outline with a pair of scissors, and then run a pencil round the margin on a piece of white paper, and you have a fairly correct outline of the ulcer. If you repeat this at certain definite intervals, say of a week, and place each outline within the other you will get a good indication of the progress made. This diagram I show you is an exact copy of a drawing taken in the way I have described.

To return now to the local treatment of the ulcer. In the first place we have an exposed raw surface, and here, as in every other



kind of surface lesion, cleanliness is essential for rapid healing. We therefore devote our attention during the first few days to repeated and frequent dressings of the part with some simple antiseptic solution. It matters little what you employ, provided you avoid all greasy preparations. Discard ointments and such like preparations as inhibitory rather than otherwise to the attainment of perfect cleanliness. You must often have seen the simple dressing we employ,—that is, a piece of boracic lint large enough to cover the ulcer, wrung out of a weak carbolic solution and then well covered by a piece of gutta-percha tissue. This is removed every night and morning, and replaced on each occasion with a fresh dressing.

As soon as the ulcer appears sufficiently clean, we proceed to treat it by systematic massage. The object we now have in view is by well-directed pressure, rubbing, and kneading to get rid of the contents of the engorged veins and lymphatics, to facilitate the absorption of the surrounding exudation, and to cause a re-entry of fresh arterial blood to the part.

In order to make clear to you the method of carrying out this process, I will proceed to demonstrate it upon this patient. The ulcer is exposed and cleaned with a little solution. A piece of jacconet is taken of sufficient size to extend well above and below the ulcer and wide enough to more than encircle the limb. The leg is raised by the nurse supporting the heel, and, standing at the foot of the bed, I take the piece of jacconet, dip the non-glazed surface in some solution, and then apply it to the ulcer with the glazed surface external. With the left hand I grasp tightly the free margins of the jacconet at the back of the leg, so causing the tissue to apply itself smoothly over the ulcer and the skin around. A small quantity of vaseline is then smeared upon the glazed surface of the jacconet, and, with the right hand spread over the anterior part of the leg, I proceed to rub upward from the dorsum of the foot, over the ulcer, to the healthy tissues of the leg above. The amount of pressure exercised depends upon what the patient can stand. The process at first is frequently a painful one, the more so if there is inflammation about the skin surrounding the ulcer. As, however, the engorgement subsides, the pain proportionately diminishes and more active massage becomes possible.

The method I have just shown you is one specially applicable for those cases where the base of the ulcer is pale and glazed-looking,

showing a sluggishness in the process of repair. Where there appears sufficient vascularity of the granulations in the floor of the ulcer, we need not massage directly over it, but devote our attention to pressing and rubbing the skin around. The ball of the thumb answers best for massaging the edges of the ulcer. But coupled with this more limited method of massaging, the foot below and the leg above should always be well rubbed upward, so as to stimulate the circulation of the whole limb.

You may take it as a good sign when you find that the congested skin around the ulcer pales on pressure, for it indicates that the blood can be pressed from the part; in other words, that as the vessels can be emptied they stand a good chance of being filled with fresh blood.

Reverting to that part of the treatment which concerns the excision of a varicose vein, you will be surprised to see how rapid sometimes is the improvement which follows in the appearance of the ulcer. It is frequently observed on the following morning, especially in those cases where the engorgement has not been sufficiently prolonged to lead to chronic thickening and induration of the surrounding tissue, and where, therefore, the removal of the obstruction to the blood-supply was all that was necessary to allow of rapid repair.

It may be asked by some one of you, and not without reason, How is the circulation carried on if some of the main return channels are removed as in venectomy? The answer is that the deep veins appear to exercise a function sufficient to compensate for this diminution in the number of the subcutaneous channels; and I may forestall by an answer another question which you might as reasonably ask, that this increased strain on the deep vessels does not cause any subsequent inconvenience to the patient. It was at one time alleged that if you removed a large vein like the internal saphena, the patient would suffer from a sense of weight and deep-seated pain in the leg owing to a compensatory enlargement of the deep veins. All I can say is that, in now a tolerably large experience of venectomy for varicose veins, I have never yet met a patient who has thus suffered, or who indeed has not, on the other hand, been much benefited by the operation.

To sum up briefly for you the advantages which I think may be claimed for the treatment of congested ulcers by venectomy and massage, I would give the following:

1. Improvement shows itself at once, and is most marked during the first week or two. The heaped-up skin edges begin rapidly to disappear and a sloping healing "blue" line is observed. The floor of the ulcer soon vascularizes and shows healthy florid granulations. The surrounding skin becomes early whitened or mottled, indicating the complete emptying of the congested venules.

2. Later, the skin around becomes more pliable and can be pinched up and freely moved on the underlying tissues.

3. The cicatrix formed is well organized and thus less liable to break down again.

4. Massage can be carried out by a nurse or by the patient's friends; and if continued for some time after healing has taken place will maintain the skin and soft parts in a healthy condition.

# CHICAGO SANITARY FLOUR FOR CERTAIN DYS- PEPTICS AND DIABETICS.

LECTURE DELIVERED BEFORE THE CHICAGO MEDICAL SOCIETY.

BY N. S. DAVIS, JR., A.M., M.D.,

Professor of Medicine and of Clinical Medicine in the Northwestern University  
Medical School, Chicago, Illinois.

GENTLEMEN,—I take pleasure in showing the Chicago Medical Society samples of a new flour, invented by a former patient of mine, C. C. Jerome, of this city. He was led to seek for his own use a flour free from the features of ordinary wheat and gluten flour, objectionable to diabetics.

The nuts from which this flour is made are subjected to high pressure, which extracts a large amount of oil from them. The cake which is thus made is ground into a fine, slightly yellow flour. It has an agreeable, bland taste. It contains no starch. Professor J. H. Long, of Northwestern University Medical School, has made a thorough analysis of both the flour and the bread made from it. The flour contains of

Water . . . . .	8.01 per cent.
Fat. . . . .	19.82 "
Albuminoids . . . . .	55.65 "
Sugar. . . . .	6.25 "
Mineral salts . . . . .	6.82 "
Fibre and other non-nitrogenous matter . . . . .	8.95 "
	<u>100.00</u> "

As very palatable and attractive food products can be made from it, it will prove a valuable article in the dietary of certain invalids. Bread and biscuits made from it do not readily ferment, as they contain no starch and very little sugar. The oil which these nuts contain, and which remains in small amounts in the flour, is peculiarly stable, not undergoing fermentation or decomposition, even in mid-

summer, when exposed freely to heat and to the air. Food prepared from this flour can be advantageously used by those dyspeptics in whom starches ferment with readiness, and by whom they cannot therefore be used.

My attention has been called to the fact that in several instances, when food prepared from this flour was used daily and for some time, mild but habitual constipation was relieved. It seems probable that the oil contained in it has a gentle stimulating influence upon intestinal peristalsis. In no case have I learned of any symptoms of indigestion, either gastric or intestinal, following its use.

The chemical composition of this bread fits it especially for the use of diabetics. Although this flour contains about six per cent. of cane-sugar, the bread when leavened with yeast contains less than one-half per cent. The sugar is decomposed, and the gases thus set free raise the dough. No article of food that is forbidden to diabetics is more keenly missed than bread. It is true that in the largest proportion of cases they need not be wholly deprived of it, but in all cases the amount taken must be limited. A safe substitute is much to be desired. I have found it impossible to permit diabetics to use so-called gluten breads, for they invariably eat them freely, and as all these breads contain from thirty-five to seventy-five per cent. of starch, patients get more amylaceous food than they can utilize. Moreover, in proportion as gluten breads are poor in starch and rich in gluten do they become unpalatable and unattractive. It is much safer to permit diabetics to use a measured amount of good wheat bread, the exact composition of which is known, than breads made of the gluten flour in the market. As this new bread contains no starch and a minimum quantity of sugar, it is a peculiarly good substitute for wheat bread for diabetics.

A considerable variety of foods can be prepared from this flour. The bread leavened with yeast is peculiarly fitted for use by diabetics. Muffins raised with baking-powder can be used with advantage by many dyspeptics, as can ginger cookies and the flour prepared as a breakfast food and eaten with cream. Pie crust, pancakes, and other articles of good quality can be made from this flour, although they are not as well adapted to the use of invalids as those already mentioned.

I have had an opportunity to watch the use of this flour in so limited a number of cases that I cannot discuss its limitations as fully

as will be possible after a more extended trial. I will only contrast the results obtained in one case of diabetes by the use, during successive weeks, of one of the best gluten flours in the market and this one. While the gluten bread was eaten my patient voided on the average one hundred and fifteen ounces of urine, which contained 1.8 per cent. of sugar. The average daily output of sugar was therefore a little more than two ounces. While he used exclusively the bread prepared from this sanitary flour he voided daily an average of ninety ounces of urine, which contained 1.3 per cent. of sugar. This represented a daily excretion of approximately one and one-sixth ounces of sugar.

## THE TREATMENT OF WHOOPING-COUGH.

CLINICAL LECTURE DELIVERED AT THE HÔPITAL DES ENFANTS.

BY PROFESSOR MARFAN, M.D.,

Professor Agrégé in the Medical Faculty of Paris, France.

---

GENTLEMEN,—As we have at the present moment a number of little patients in our pavilion with whooping-cough, I shall devote this lecture to that complaint, and more especially to its treatment. Although I shall not have anything very new to tell you about the disorder itself, the same cannot be said of its treatment, as new remedies are constantly being found for whooping-cough, although no one of them has as yet proved absolutely satisfactory.

You are all aware that this disorder is a specific catarrh of the respiratory tract, localized to the nose, larynx, and trachea, and that it is handed over from child to child by contagion only; it is therefore unquestionably caused by some parasitic agent that develops on the mucous membrane of the nose, larynx, and trachea, and brings on the characteristic fits of coughing. I believe that the idea of a specific parasite is now accepted by everybody, and we are therefore authorized in seeking for some specific treatment.

Is there a specific treatment for whooping-cough? A great deal of research has been devoted to this point, and every now and then some physician announces to the world the discovery of a standard treatment that will radically check the complaint, or that will at least noticeably diminish its inconveniences. Let us briefly consider some of these statements.

This disorder can be divided into three periods:

1. The initial period, characterized by fever and by catarrhal conditions with no special features, lasting about a week.
2. The period during which the disease is in full force, with the familiar coughing fits, but without fever, and lasting at least a month.

3. The period of decline, variable as to length, but usually lasting about ten days.

During the first period we are absolutely incapable of making the diagnosis; it is not, therefore, at that period that we can test a specific treatment.

If a specific remedy exists, it ought certainly to lessen the symptoms of the second period; unfortunately, we must frankly confess that at the present time there is not a single chemical compound on which we can bestow the title of a specific; neither the oxymel scilliticum,<sup>1</sup> sulphate of quinine, creosote, or guaiacol, nor local applications of resorcin to the pharynx can be considered a specific remedy for whooping-cough.

From this, we are by no means to think that we are powerless in the presence of this disease. We still have a symptomatic treatment to use, although it is a somewhat complex one.

In prescribing such a treatment we must first ask ourselves what the dangers are to children suffering from whooping-cough. These are of two kinds. The first depend on the number and violence of the coughing fits, the second on the possibility of the occurrence of secondary infection of the bronchial tubes.

The special character of the coughing fit in this disorder is its violence and rhythm. It begins by a series of exaggerated spasmodic expirations, which are followed by a period of inspiration, deep and wheezy; a second series of spasmodic expirations then occurs, also followed by a period of inspiration. Then, after a moment of rest which is invariable but always brief, a new series of expirations and inspirations take place, the entire attack being composed of a series of paroxysms in which can be distinguished two or three series of expirations and inspirations. The truth of this can be readily verified by means of Marey's pneumograph.

There is nothing that can be mistaken for an attack of whooping-cough, unless it be the kind of cough sometimes noted in tumors of the mediastinum, where pressure is brought to bear on the pneumogastric nerve.

Together with the characteristic cough we find very pronounced venous stasis, intense swelling of the jugular veins, congestion of the face, cyanosis of the extremities, etc. This condition may even go so far as to rupture vessels, cases having been reported of epis-

---

<sup>1</sup> Codex Médicamentarius (French Pharmacopœia), Paris, 1884, p. 462.



taxis, purpura, meningeal hemorrhage, and cerebral hemorrhage; the conjunctiva has been known to bleed and the patient drop tears of blood. There is sometimes œdema of the lower limbs or of the glottis, and puffiness of the face. Consequently we find venous stasis that ends either in hemorrhage or œdema.

While a child is having a coughing fit of this sort the volume of air taken in in an inspiratory movement may overtax the elasticity of the lungs and give rise either to pneumothorax or emphysema of the vesicles, mediastinum, or subcutaneous tissues. Another consequence of this accident may be increased tension in the abdominal cavity, producing vomiting. It is easy to explain the occurrence of vomiting in this complaint, as when the period of deep inspiration begins the tension in the thoracic cavity is diminished, whereas in the abdomen it is increased. In other cases involuntary stools, or micturition, may occur, or else prolapsus of the rectum.

This will give you an idea of the more or less serious accidents that may be the result of the frequent or violent coughing fits in this disorder. Furthermore, we have also to bear in mind the fact that a secondary infection of the respiratory tract is always possible, and is one to which the little patient would quickly succumb, an easy victim, should the child be found exhausted and in a cachectic condition from the coughing fits.

We have therefore two great therapeutical indications to consider:

*First*, to lessen the number and intensity of the attacks.

*Secondly*, to prevent, or combat, the secondary bronchial infection.

When we examine such a patient with the rhinoscope and laryngoscope we only see that the mucous membrane lining the nose, larynx, and trachea is red, and that it secretes a limpid, sticky liquid. These are the two dominant characteristics of the mucous membranes in whooping-cough: redness and viscous secretion.

But on this catarrhal condition a secondary infection of the strepto- or staphylococci may graft itself. These children, in addition to their coryza, develop ulcerations in the mouth, which then become covered with bloody crusts. When the latter fall they leave diphtheroid patches on the lips, gums, and under the tongue. This is the first stage of an autoinfection due to contagion.

When this condition is discovered there is fear that it may go

down to the bronchial tubes and set up bronchitis, which is extremely common. This complication may be unimportant, superficial, and manifest itself to the ear by a few sonorous rhonchi only, or it may become diffuse and manifest itself by abundant, loud rhonchi, which may usher in a bronchopneumonia. If the child is not too young you know that you must at once apply the treatment of hot baths.

To return now to our two therapeutical indications. To diminish the intensity and number of the coughing fits it is probable that every spasmodic remedy known has been tried. I shall not weary your patience by going over the very long list of drugs that have been abandoned, but shall attempt to make a more definite impression on your minds as to what substances you are to use by telling you that for practical purposes you need only remember three remedies of this class,—belladonna, antipyrin, and bromoform.

Belladonna, which was first brought into prominence by Trousseau, is no longer given according to his formula, which was half a centigramme (about one-twelfth of a grain) each of extract of belladonna and of powdered belladonna leaves, for one pill. The dose was one pill for very young children, and two for children over four years of age; the dose was given each morning on an empty stomach for one or two weeks, and if at the end of that time the coughing fits were as frequent as ever, he doubled the dose. Whatever may have been said to the contrary, he kept a very close watch on the effect of the remedy on his patients. His method, however, was given up mainly on account of the difficulty experienced in getting children to swallow pills.

Nowadays the two preparations generally used in France are the syrup and tincture of belladonna. Cadet de Gassicourt prescribes as follows:

R Syrup of belladonna, 50 gms. or  $\text{℥}$ iss;  
Syrup of tolu, 150 gms. or  $\text{℥}$ ivss.  
Sig.—As directed.

Half a teaspoonful should be given twice a day to young children, increasing the dose gradually. For children over seven, the initial dose should be a teaspoonful.

M. Roger advises the following formula:

R Syrup of belladonna, 50 gms. or ℥iiss;  
 Syrup of valerian, or ether,  
 Syrup of digitalis, aa 25 gms. or ℥vi. M.  
 Sig.—As directed.

Under two years of age, give half a teaspoonful twice a day; increasing the dose gradually to twice this amount. From two to five years of age you can go to four teaspoonfuls a day, the maximum dose, and over five years, to a maximum of six teaspoonfuls. The digitalis is added because these patients have a rapid pulse, and experience has shown the usefulness of this addition.

Jules Simon uses the following preparation:

R Tincture of belladonna,  
 Tincture of aconite root, aa 10 gms. or ℥iiss.

This must always be given in small and progressive doses, so that its action can be watched; one-quarter to two drops for children less than two years of age, and from one-half to six drops for older children.

My own preference is for this preparation:

R Syrup of belladonna, 20 gms. or ℥ss;  
 Syrup of tolu, q. s. ad. 100 gms. or ℥iiss. M.  
 Sig.—As directed.

Below two years of age, one to two teaspoonfuls a day; from two to five years, three teaspoonfuls a day; after five years, five teaspoonfuls a day.

Bear carefully in mind that this is the *initial* dose, which must be gradually increased to twice the amount, often before the coughing fits are lessened. You need not decrease the dose unless the patient shows signs of intolerance, such as dryness of the mouth or throat, or dilated pupils.

With the use of belladonna we succeed in some cases in controlling the paroxysms of whooping-cough, but it is only right that I should tell you that we often get signs of intoxication before the coughing fits have been affected at all. This is why a number of medical men have turned in preference to the second of the remedies I advised you to remember,—antipyrin.

This drug should always be given in solution; children stand

it in a remarkable manner, and it easily lessens the number and intensity of the paroxysms. The formula I use is as follows:

R Antipyrin, 8 gms. or  $\mathfrak{z}\text{i}$ ;  
 Syrup of orange flowers, 20 gms. or  $\mathfrak{z}\text{ss}$ ;  
 Distilled water, 100 gms. or  $\mathfrak{z}\text{iiss}$ . M.  
 Sig.—Use as directed.

One teaspoonful would represent two grains of antipyrin, a dessertspoonful four, and a tablespoonful eight grains. Under two years I give from two to fifteen grains. To older children from one to three grammes (fifteen to forty-five grains), as a maximum dose. Always begin by a small dose, and do not be timid about increasing it. In four or five days you will find that the paroxysms generally grow less violent and frequent. The remedy can be continued for a long time. When children cannot be made to take this preparation, the remedy can be given as an enema, but the antipyrin should then be dissolved in only a little water, twenty centigrammes or four grains in forty grammes or an ounce of water, for instance. The bowels should first be emptied, and the remedy given with a long canula, night and morning.

When bronchopneumonia exists as a complication of the complaint, antipyrin should not be used on account of its depressing action on the heart.

It is possible that in the third remedy to which I called your attention we have one that is destined to supersede both of those of which we have spoken.

Bromoform is a limpid liquid, with an agreeable odor, not unlike that of chloroform. It is soluble in glycerin and in alcohol. Stepp, of Nuremberg, claims that this drug lessens the number and intensity of the coughing fits, prevents secondary infection, and shortens the duration of the case. He gives bromoform in an alcoholic solution, by drops. The formula I have finally adopted is as follows:

R Bromoform, 48 drops;  
 Oil of sweet almond, 15 gms. or  $\mathfrak{z}\text{ss}$ .

To this add

Gum arabic,  
 Syrup of bitter orange,  $\mathfrak{aa}$  15 gms. or  $\mathfrak{z}\text{ss}$ ;  
 Distilled water, q. s. ad 120 c.c. or  $\mathfrak{z}\text{iiss}$ . M.  
 Sig.—Use as directed.

When this is properly compounded, each teaspoonful contains two drops of bromoform. Below six years of age, give as many times four drops as the child is years old; thus, to a child three years of age give twelve drops; below the age of six months, begin with one drop; from six to twelve months, one or two drops; after six years of age, twenty drops.

These are all initial doses, and should be increased two drops per diem. During the first two or three days it sometimes happens that the number of paroxysms appears to increase, and that they become more violent, but by the third or fourth day there is a noticeable improvement, which becomes more marked later on.

Bromoform does not succeed in all cases; in fact, it sometimes fails completely, and you must then fall back on antipyrin. In other instances the three remedies all fail,—belladonna, antipyrin, and bromoform; in that event a combination of antipyrin with belladonna may succeed.

R Antipyrin, 8 gms. or grs. xlv;  
Syrup of belladonna, 20 gms. or ʒv;  
Distilled water, q. s. 100 gms. or ʒiii. M.  
Sig.—To be given as directed.

To sum up, let me tell you that I now begin to treat a case of whooping-cough with bromoform; if that fails, I try antipyrin, then belladonna, and finally fall back on a combination of the last two. At the present time bromoform appears to be the remedy that succeeds oftenest, and I think it is destined to render us signal service. It is a good general rule to subdivide the daily dose of an energetic remedy; this should not be forgotten with bromoform, and the daily dose should be divided into three parts and given at equal intervals.

Some writers have called attention to certain cases of intoxication, said to have followed its use, in which they claim to have observed a condition resembling coma. For my part, I have never yet seen anything of this nature; but should the remedy at any time produce a marked state of somnolence, it had better be given up.

As regards the second great indication in treating whooping-cough, the prevention of secondary infection, it is advisable to free the bronchial tubes from the secretions that accumulate in them in order to avoid the development of capillary bronchitis.

For this purpose many physicians prescribe emetics, some in doses that simply cause nausea, others in doses which produce vomiting. Jules Simon, for instance, is in favor of causing his little patients to vomit about once in five days. I must confess that I do not look with favor on this systematic mode of procedure. In the first place, the efficacy of an emetic is open to question; again, they exhaust the patient's strength; and lastly, they have been known to cause sudden death by asphyxia and syncope in very young children in a weak or cachectic state.

I am in the habit of using the various balsams in the place of emetics, as they not only disinfect to a certain degree the respiratory passages, but are useful as expectorants at the same time. In ordinary cases the atmosphere of the room in which the little patient lives can be saturated with balsamic vapors by means of a platinum vaporizing lamp, or any simple process, such as placing, three or four times a day, over the flame of a night-lamp, a certain quantity of the following balsamic mixture, and allowing it to evaporate:

R Essence of thyme,  
 Essence of eucalyptus,  
 Essence of turpentine, aa 10 gms. or ʒss;  
 Alcohol, 250 gms. or ʒiiss;  
 Water, 750 gms. or ʒivss. M.  
 Sig.—To be used as directed.

If this external administration of the balsamic mixture does not suffice, they can be given internally. Benzoate of sodium, for instance, can be prescribed, one-half to one gramme (7–15 grains) per diem to children less than one year old. To older children you can give terpene, or terpinol, which are excellent remedies, though they can only be dissolved in an alcoholic preparation. Creosote has also been used for this purpose, administered by the rectum.

In addition to the two leading indications in the treatment of whooping-cough, and of which we have spoken, there is a third which is also of importance,—careful supervision of the hygienic conditions of these patients. This should be attended to down to its minutest details. So far as possible, the child's room should be well ventilated, airy and sunny, which will not harmonize with the preconceived notions of many parents. It is well to move the patient into a different room every few days, or even to have different rooms for the day and night. Without our knowing exactly why, this practice very often produces marked improvement.

The point is always raised whether these patients are to be allowed to go out. No *a priori* answer can be given to this question, as it depends altogether on circumstances. Jules Simon forbids their going out systematically, and shuts them up hermetically until they recover; others, on the other hand, allow them to go out. For my part, I allow myself to be guided by circumstances. If the weather is fine, sunny, and warm, I let them go out, particularly if the paroxysms are not more than of average frequency or intensity. If the fresh air and exercise does not increase the paroxysms I continue to let them go out, but, on the contrary, eventually I have them kept in the house.

You must not forget to tell the people about the patient what to do during the paroxysm: the child should be at once seated, and the forehead should be supported by some one's hand until the attack is over.

Let me now say a few words about the ordinary complications of this complaint. When the paroxysms are followed by vomiting, the best plan is to get the child to eat again at once. Another empirical but excellent means is to give weak, cold coffee, with little or no sugar in it,—from a teaspoonful to a tablespoonful, according to age, immediately after eating, except in the evening when it might interfere with sleep.

I cannot undertake to-day to describe to you the treatment of broncho-pneumonia occurring as a complication of this disorder, but I may mention some of the precautions to be taken to avoid it.

The usual pathogenic microbes of this complication are, as you know, the pneumococcus and streptococcus which are commonly to be found in the nose and mouth. It is consequently desirable to do what you can to disinfect these cavities, in order to lessen the virulence of these micro-organisms, which is probably increased during whooping-cough. For the nose you should have a lump the size of a pea of the following preparation put in each of the child's nostrils three or four times a day, the head being held well back:

R Boracic acid, 6 gms. or  $\text{ʒiiss}$ ;  
Menthol, 0.05 centig. or gr.  $\frac{1}{20}$ ;  
Vaseline, 80 gms. or  $\text{ʒviiss}$ . M.  
Sig.—For local use.

The preparations of resorcin are also excellent, oil of vaseline

and resorcin in a strength of one to fifty or one to twenty-five. For the mouth, rinse it well each time after eating, with,—

**R** Carbolic acid, 1.50 gms. or grs.  $\text{xx}$  ;  
 Thymol, 0.10 centig. or gr.  $\text{iss}$  ;  
 Distilled water, 500 gms. or  $\text{℥xvss}$  (or  $\text{Oι ℥iss}$ ). **M.**  
 Sig.—Use as a mouth-wash.

The reason why I lay such stress on this care of the mouth and nose is that ulcerative rhinitis and stomatitis are so common with these patients. These ulcerations can be found quite as often on the lips, and back of them, as under the tongue; you will also find that bronchopneumonia generally occurs with patients who present these lesions of the mouth and nose. I may add that when these lesions do not disappear with the use of the above-mentioned preparations, I do not hesitate to cauterize them with a one to thirty solution of nitrate of silver, which is certain to heal them up.

When convulsions occur after or during the paroxysms, you should give the child ether or chloroform to breathe on a handkerchief, to be followed by bromide of sodium or chloral in a draught or as an enema.

Finally, when a child suddenly stops breathing or has a syncope during a paroxysm, you should lose no time in bringing every means to bear to restore the respirations,—flagellation, artificial respiration, electrization of the phrenic nerves, and rhythmical traction on the tongue.

The details I have given you in this lecture are only connected with the treatment of a fully developed case. During the preceding stage the prognosis cannot be made, and the treatment can only be that of bronchitis. In the third stage, when the catarrhal bronchitis depends on emphysema and shows a tendency to become chronic, you will get good results from counter irritation over the chest with iodine or croton oil; but one of the very best means to put an end to the complaint at this stage is a complete change of air. Therefore, if, in spite of every plan of treatment, the child still continues to cough, the best course to be followed is to prescribe a course of treatment at a suitable mineral-water establishment, such as the Eaux Bonnes, Cauterets, or the Mont Doré.



## PLACENTA PRÆVIA: ITS DANGERS AND TREATMENT.

BY J. W. BALLANTYNE, M.D., F.R.C.P.E., F.R.S.E.,

Lecturer on Midwifery and Diseases of Women in the School of the Royal Colleges, Edinburgh; Examiner in Midwifery in the University of Aberdeen, etc., Scotland.

---

GENTLEMEN,—Placenta prævia is a complication of labor the occurrence of which is dreaded, and rightly so, by the practitioner, for it brings with it not one but many dangers. We naturally think, first, of the immediate evil effects which may follow the hemorrhage which occurs as a consequence of the low position of the placenta in the uterus; but this is only one of the risks associated with the anomaly. What these risks are I hope to make clear to you, and thereafter to describe the methods of treatment which seem best calculated to counteract or annul them. The success of any method of treatment must under such circumstances be judged not in respect to one of the dangers, but to all; for one sort of management may very adequately meet one risk, and yet be ineffectual, or worse than ineffectual, with regard to the others.

### DANGERS TO THE MOTHER.

The *first*, the most apparent, and the most clamant danger is the bleeding from the vagina which inevitably occurs sooner or later in the pregnancy in which the placenta is prævia. The patient is usually a multipara, sometimes also a pluripara; she is within a month or two, or even three, of the full term of her pregnancy; and she has in no way been over-exerting herself or undergoing injury. Suddenly, without warning, often in the night and not uncommonly during sleep, hemorrhage of a more or less terrifying kind sets in from the vagina. The bleeding may be so torrential as to cause immediate danger of death; and the woman may die, especially if her abode be far from that of her doctor, with an undilated os uteri and in the absence of skilled assistance. This lamentable result, how-

ever, is rare, and probably occurs only when the placenta prævia is of the central or total variety. This hemorrhage is due to the partial separation of a placenta situated in the lower part of the uterus, in the portion which dilates during labor. It is now generally admitted that it is largely uterine in origin, but that a small amount of blood may come from the placenta itself is also not to be denied; the torn-across vessels of the uterine wall are, however, much the more important source. When it occurs a month or two before the full term of pregnancy, it may cease after some minutes and recur some days or weeks later; but when it takes place near the close of gestation, uterine contraction commonly supervenes, the bleeding becomes more marked, and, if no help is at hand, matters may be very serious for the patient. Even when the bleeding sets in at the seventh month, labor may commence and the necessity for immediately delivering the woman arise. That this symptom is not due to carcinoma of the cervix is usually easy of determination, for a vaginal examination will fail to disclose the characteristic cancerous changes and will reveal the presence of a soft structure, the placenta, lying in advance of the presenting part, which will be indistinctly palpable. If the os be partly open, the detection of the placental mass with its "wormy" vessels in the central variety, or of the placental margin and attached membranes in the marginal or lateral variety, will be easy. The danger from the hemorrhage will vary in degree in different cases: it will be great in the central form, less in the marginal, and still less in the lateral type; it will be much greater if no skilled assistance is at hand; and it will of course be greater if the patient has been already weakened by its frequent recurrence. We may judge of the degree of danger partly from the history of the hemorrhage which the patient and her attendants give us, partly from the amount of the bleeding which is taking place under our own observation, and partly from the results of vaginal examination and the consideration of the general symptoms which are present. When the bleeding has been comparatively slight, the patient will show some pallor, may have sympathetic vomiting and impaired vision, and may feel faint; but in the cases where the blood-loss has been great there will be actual fainting, great pallor, rigors, even convulsions, vomiting, and incoherent muttering, along with marked dyspnœa. The danger is lessened by the supervision of strong labor pains, especially if the full term of pregnancy has been nearly

or quite reached, and the natural rupture of the membranes, which occasionally occurs, will still further diminish it. As the head or other presenting part gets fairly into the dilated cervical canal, the bleeding will probably stop, and then, save in cases of central placenta prævia, much of the risk is at an end.

The *second* danger is also due to hemorrhage, but in this instance it is hemorrhage after the birth of the infant. It may arise in two ways,—from laceration of the cervix due to too rapid extraction of the fœtus, or from uterine inertia. In the first case it will immediately follow the birth of the infant, while in the second it may occur before the placenta is removed or after it. An example of the first of these two types I met with in the early part of 1890. I saw the patient, who had a central placenta prævia, in consultation. The bleeding was very alarming, and in order to control it the cervix was quickly dilated, the placental tissue pierced by the hand, and a fœtal foot drawn down. There was still some bleeding, and, possibly in response to the openly expressed importunity of the patient's husband, who was also a medical man, the infant was dragged through the cervix with a celerity not compatible with safety. The result was a fairly deep cervical tear and a most alarming hemorrhage, during which the patient very nearly died. The bleeding was with great difficulty arrested, and a fair recovery followed. The case produced a great impression on my mind at the time, and I have ever since kept this danger fully in view when treating cases of placenta prævia. The bleeding may also be from the uterine body from inertia of that organ. Possibly on account of the rapid delivery of the infant and placenta, the uterus may fail to retract, its interior becomes filled with blood, and we are at once face to face with the added danger of post-partum hemorrhage. Even before and during the removal of the placenta there may be bleeding, for the placenta which is prævia is often also adherent, and is not uncommonly of irregular form. In some instances, further, a succenturiate lobe exists and may be left in utero, and so cause yet more bleeding. The membranes also may be adherent to the uterine wall.

The *third* danger is septic infection of the patient. This is doubtless specially predisposed to by the weakening effect of the hemorrhage; but it is also largely the result of the prolonged and often complicated means of treatment adopted to arrest the bleeding, and of the instrumental or manual manœuvres necessary for

the version and extraction of the infant and for the removal of the placenta. It may also result from the leaving behind of placental *débris* or membranes in the uterus. It may take the form of acute sepsis and prove fatal in a few days, the weakened patient succumbing rapidly to the action of the poison. The worst case of puerperal sepsis I have seen followed extensive hemorrhage, and but for vigorous curettage of the uterus would, I believe, have terminated fatally. On the other hand there may be a chronic infection of the uterine mucosa leading to the long succession of morbid phenomena associated with the development of endometritis, endosalpingitis, ovaritis, and pelvic peritonitis and cellulitis. Between these two forms of infection lies phlegmasia alba dolens, or "white leg." Notwithstanding all my care this developed in one of the cases of placenta prævia which have been under my charge; but it may be mentioned that the patient had had, in a former pregnancy, not accompanied by placenta prævia, this same puerperal complication. The recovery was thus rendered tedious, but was ultimately complete. Among the less direct and less frequent dangers of placenta prævia may be mentioned puerperal insanity, puerperal pulmonary thrombosis, and uterine subinvolution.

#### DANGERS TO THE INFANT.

Thus far I have spoken only of the dangers to the mother; but with the mother suffers *the infant*, and the risks that she runs are shared in by her unborn child. The great danger to which the foetus is liable in cases of placenta prævia is antenatal death, and there seems to be good reason to believe that this fatal result, when it occurs, is due not so much to loss of foetal blood as to imperfect aëration of it. There is true foetal asphyxia rather than foetal syncope from anæmia. The danger to the foetus will therefore be increased *pari passu* with the augmented imperfect functioning of the placenta as a blood-purifying organ rather than with increased blood-loss. In this way may be explained some apparently anomalous cases in which the hemorrhage has been very great, and yet a living infant has been born to an almost moribund mother. Foetal death from asphyxia may occur several days—even two or three weeks—before the mother is delivered, and this in its turn is an additional source of danger to the mother and still further increases her liability to sepsis. When the infant escapes antenatal death, it may yet

perish at or soon after birth, either from injuries received during the manipulations undertaken on behalf of the mother or from asphyxia neonatorum resulting from the pre-existing unsatisfactory conditions *in utero*. This danger is greatly increased by the frequent occurrence of premature labor, by the abnormal presentations so often found in association with placenta prævia, and by the prolapse of the cord, which is a common and most troublesome complication. Even when a living infant is brought into the world its further existence is by no means assured, for it is often weakly even when not premature, and it has to be nourished artificially or nursed by an anæmic or otherwise unfit mother. Truly we have here a vicious circle of a most peculiarly vicious type. One danger brings into evidence another, and the other reacts upon and intensifies the first one. It need scarcely be wondered at that cases of placenta prævia are dreaded by even the most skilled obstetrician, and that their tragic effects are long remembered by the patient, her friends, and her medical attendant. In the last case of this complication of labor which I met with (in June, 1897) a XV.-parous woman deplored the successive birth of four dead infants at or nearly at the full term; in two of these the cause had certainly been placenta prævia, and in the other two it may have been so; and in her fifteenth pregnancy the now so familiar and so greatly dreaded hemorrhage appeared again at about the eighth month. On my arrival I found the woman naturally much depressed and most anxious, both about herself and about the fate of her infant. The placenta prævia fortunately was of the lateral variety, and I succeeded by means immediately to be described in delivering the patient of a female infant, which was with difficulty resuscitated and unfortunately died a week later. The mother made a good recovery; nevertheless her whole history was a tragic one, and the loss of infantile life, brought about directly or indirectly by the low implantation of the placenta in these five labors, was as bad as it could be, amounting in fact to one hundred per cent. The case had a special interest from the fact that in three at least, and possibly also in five, successive pregnancies the placenta had been prævia. The woman's earlier confinements had all been normal and uneventful.

*Treatment.*—The treatment of placenta prævia is never a simple matter. The condition is too complex to permit of the laying down of any hard and fast rules for treatment. The anomaly is the cause

of so many dangers both to mother and child that nothing but a wide consideration of all the circumstances of the case, an elastic plan of procedure which can be modified to meet unexpected developments, and a resourceful dexterity in the application of operative measures are sufficient to cope with them. Herein lies the explanation of the diversity of opinion regarding the method of managing cases of placenta prævia. One obstetrician rarely meets more than a few cases in the course of several years, and the treatment he adopts and finds successful in these might be quite inadequate in other instances. He naturally enough extols his method, while others, equally naturally, are sceptical regarding its advantages because they have tried it and found it wanting in probably quite a different kind of case.

There is, as I have already said, a vicious circle of occurrences in the course of a case of placenta. The first indication for treatment is to cut into this circle at some point, and the point usually chosen is the arrest of the hemorrhage. Many plans have been suggested, and our problem is, in any given instance to choose that which, whilst it must be adequate for the stoppage of the bleeding, will also be simple and unlikely to increase the other dangers which the anomalous implantation of the placenta gives rise to. The more complicated the method the greater will be the risk of sepsis and of its subsequent morbid developments.

In some measures an indication as to the best line of treatment is found in the diagnosis of the variety of placenta prævia with which we have to deal. If we can feel the placenta set fairly over the internal os, then more energetic measures will probably be necessary than when it is lateral or marginal. Even before dilatation of the cervix we may suspect the variety which is present by noting the date of onset of the hemorrhage: bleeding at the seventh month usually indicates the central type, at the eighth month the lateral, and at the ninth the marginal. When bleeding supervenes late in pregnancy, when it is not excessive, and when labor pains come into action with steadily increasing force, we may with a large degree of safety wait and watch. But it is well to bear in mind that the watching is more important than the waiting. We must not leave such a case, but must remain in close attendance. In many cases, as in two with which I have had to do, the dilatation of the cervix is accompanied by the rupture of the membranes and the descent of

the presenting part, and as a consequence cessation of the bleeding occurs and much of the risk is over. We may or may not require to use forceps, but in such cases I believe in abstention from active interference united with the closest observation of events and the strictest antiseptic precautions. The escape of the liquor amnii allows the uterus to contract upon its contents, and the descent of the presenting part pushes to one side the placenta and its separation for the time ceases. If labor pains do not supervene, and if the bleeding is comparatively slight and intermittent, then again the policy of reasoning, observant, and prepared inactivity will be the best. The bad symptoms may pass away and the pregnancy go on to the full term, and so a better chance be given to both mother and infant. Let me, however, again warn you that the inactivity must be the result of the knowledge gained from a close scrutiny of all the facts of the case, and not be the expression of ignorance and want of appreciation of danger. Believe me, it is often more difficult to refrain from interference than to undertake it *when we fully realize the situation with which we are face to face*; watchful waiting is less easy than premature activity.

When, however, the hemorrhage is alarming, either by reason of its amount or the frequency of its recurrence, then action is indicated. If labor has started, the cervix being partly or wholly dilated, and the membranes ruptured, then it simply remains for us to choose our plan of procedure, always bearing in mind the dangers, maternal and foetal, which exist alongside of the immediate risk of hemorrhage. If it be possible to get past the presenting placenta, then it may be best rapidly to apply forceps to the head and extract. If, however, the breech or shoulder or face present, then turning will be indicated, and the leg of the foetus drawn down into the cervix will stop the hemorrhage and expedite further dilatation. The version in such cases will doubtless be podalic. When the placenta is situated centrally and when it is impossible to get past it, then it is good treatment to pass the hand right through it. It will then be difficult for the majority of obstetricians to refrain from immediate podalic version; but it is just possible that even here the right thing to do may now be to wait, especially if the cervix be not fully dilated. This line of treatment, which practically is equivalent to rupture of the membranes through the placenta, has of late been extolled by G. Fieux.<sup>1</sup> One of the cases which he puts upon record seems to

---

<sup>1</sup> G. Fieux, *Ann. de gynéc.*, xlvii., p. 97, 1897.

show that the escape of the waters may temporarily be sufficient to check the bleeding and so give valuable time for fuller dilatation of the canals. But, again, the obstetrician's watchfulness must not relax under such circumstances, and he must be prepared to draw down a foetal leg should hemorrhage persist or recur.

A much more difficult group of cases is that in which the hemorrhage is severe and the os undilated. In such circumstances many recommend vaginal tamponade. I may be wrong, but I have not been able to regard this procedure with equanimity, and I have not had courage to put it into practice. In considering its merits it must be borne in mind that a simple plugging of the vagina is not what is intended, but a most complete filling of it with a large number of pledgets,—some authors say a "hatful,"—till the whole pelvic cavity is filled therewith. This manœuvre demands both time and a certain amount of dexterity which is truly not inconsiderable. Further, the hemorrhage, if it continue, is now no longer visible, and is on this account none the less dangerous, while to unpack the vagina, if necessity should arise, will consume many precious minutes.

But if we refuse the vaginal tamponade, what treatment are we to adopt? The line of interference to which I lean and which I have followed with success is gradual dilatation of the cervix (there may be cases in which *accouchement forcé* is needed; I have not met with such) followed by rupture of the membranes, and further dilatation of the cervix and lower uterine segment by the Champetier de Ribes bag. Thereafter forceps or turning may be used as circumstances seem to indicate. The primary cervical dilatation may be done with the fingers; if greater speed be desired, the Ribes bag may be used; and then either with the fingers or with a sharp instrument the membranes are ruptured and the waters allowed to escape. Even when the membranes cannot be directly reached on account of the central insertion of the placenta, it is good treatment to perforate the presenting after-birth. In a very remarkable case reported by Fieux<sup>1</sup> this procedure (*i.e.*, rupture of the membranes) may be said to have occurred naturally, for the waters escaped during the sixth month of pregnancy. Nevertheless the pregnancy continued, and a living child was delivered seventy days later without any return of the hemorrhage. Fieux, therefore, suggests that after rupture of the membranes with a slightly dilated os, and at any time

---

<sup>1</sup> G. Fieux, loc. cit.



short of the full term, it may be well to pause in our interference and give the pregnancy a chance of going on. It does not seem that the loss of the liquor amnii constitutes a danger to the fœtus; probably the fluid is soon secreted again; certainly the gain in time is an advantage to the fœtus, which thus escapes the dangers associated with premature birth.

If, however, the rupture of the membranes is not followed by cessation of hemorrhage, then I introduce the Champetier de Ribes dilator, taking especial care by preliminary vaginal douching and cleansing of the instrument and hands that no risk of infection is incurred. The bag may be gradually distended according to the amount of resistance, and usually labor pains soon supervene, until finally the dilated bag is expelled into the vagina. I then introduce my hand and note the presentation. In the case of recurrent placenta prævia already referred to I found that the cord had prolapsed; and this is the only objection that I have to the use of the Champetier de Ribes bag. I believe it does slightly increase the risks of funic prolapse; but on the other hand, we have to consider the full dilatation of the cervical canal and lower uterine segment which it accomplishes. Should the cord prolapse, then, if it can be done with comparative safety to the mother, the immediate extraction of the child by forceps or version should be carried out. If the child be already dead, of course haste will be unnecessary.

I believe that the lines of treatment above laid down will best meet the various dangers, both immediate and remote, maternal and foetal, of placenta prævia. It must always be borne in mind that the birth of the infant does not end the risk of the case. Third-stage and post-partum hemorrhage must be guarded against, and in the puerperium too great precautions against sepsis cannot be taken. Since the infant is often prematurely born, we must treat it as such, and the incubator and a wet-nurse may be necessities if its life is to be saved.

Many of the methods of treating placenta prævia, from the administration of ergot to abdominal hysterectomy as recommended by Lawson Tait, I have not touched upon. I have simply endeavored to put before you the plan I have myself followed, and which has in a series of five cases resulted in no maternal mortality and in the death of one fœtus. The guiding principle has been watchful waiting and a resort, if necessary, to the simplest form of interference which will best meet not only one, but all the dangers of placenta prævia.

**A CASE OF TYPHOID FEVER COMPLICATED BY  
INTESTINAL HEMORRHAGE AND MENINGITIS  
DURING A RELAPSE, WITH REMARKS ON  
THE TREATMENT.**

**BY HERMAN D. MARCUS, M.D.,**

Late Lecturer on Materia Medica and Therapeutics at the Medico-Chirurgical  
College.

And

**JUDSON DALAND, M.D. (University of Pennsylvania), Philadelphia,**

Instructor in Clinical Medicine and Lecturer on Physical Diagnosis in the University  
of Pennsylvania; Assistant Physician to the Hospital of the University  
of Pennsylvania; Professor of Clinical Medicine in the  
Philadelphia Polyclinic; Fellow of the College  
of Physicians of Philadelphia.

---

N. H., seventeen years old, a girl of excellent physique, about five feet ten and a half inches tall, weighing one hundred and fifty-five pounds, was first seen on the 22d of August, 1896, for the first time, complaining of headaches, dizziness, and sharp shooting pain under the right arm, circling around the waist; gaseous eructations, and fulness of the stomach after eating. She had a very good appetite, and the bowels were regular. The headaches were located especially above the eyes, extending from the parietal bones to the occiput. The family history disclosed very little except that some aunt or uncle had died of tuberculosis. The immediate members of her family are very healthy and all of large build.

A diagnosis of anæmia was made and appropriate treatment prescribed. The result of the treatment was exceedingly satisfactory for about three months, at which time the girl returned with symptoms similar to the ones above mentioned, and, in addition, with a complaint of constant drowsiness, and the statement that sleep was at no time refreshing. Dysmenorrhœa seemed very severe, especially after the flow was fully established. Tonic treatment was

given, and the girl, after a course of two months, regained her former health. Since that time she had recurrences of these attacks at repeated intervals until May, 1897, when I was called to the house, and found the girl suffering from intense pain over the occiput, extending to the nape of the neck. The temperature, pulse, and respiration were fairly normal, the slight rise noticeable in each being accounted for by the exceeding nervousness and the fact that the girl had been suffering from this intense headache for two days before calling me in. The tongue was heavily coated and the bowels somewhat constipated. No other symptoms were noticed. A prescription was ordered consisting of—

Calomel, gr.  $\frac{1}{2}$  ;  
Salol, gr. ii ;  
Sodium bicarb., gr. ii,

to be taken every hour, and for the headache a bromide mixture was prescribed.

On calling the following day, a slight rise in temperature was noticed, the headache was persistent, and the bowels still inactive. Large doses of sulphate of magnesia were given, and with it a capsule consisting of one grain each of sulphate of quinine, salol, and acetanilide every two hours. Up to this time the diagnosis was left open, owing to the fact that, on thorough examination, both subjective and objective, no symptoms referable to any special disease could be elicited.

On returning the following day and finding that the headaches still persisted, and in fact, that their intensity had increased, especially between two and six in the afternoon, associated at the same time with a very marked rise of temperature,—namely, up to 103.4° F., gradually abating until between six and seven the temperature became normal or nearly so,—the question of malaria was considered. Quinine in doses of three grains every two hours, and ten grains at twelve o'clock noon were ordered, and the calomel treatment continued. But on returning the following day, not only was no improvement noticed, but the headache had become so severe that the slightest noise, or the slightest exertion of the patient, such as stirring the head, moving about in bed, or even a ray of light, would intensify the pain to such a degree that the patient would cry aloud. A capsule was then prescribed consisting of—

Monobromate of camphor, gr. ss ;  
 Citrated caffeine, gr. i ;  
 Acetanilide, gr. ii ;  
 Sodium bicarb., gr. ii,

to be taken every two hours.

The tongue still being coated heavily and the bowels not acting satisfactorily, the calomel, which had been discontinued for a day, was again prescribed. No amelioration of the headache was noticed, and, in fact, the condition of the patient became quite alarming, as anorexia was added to the other symptoms.

Up to this date—May 19—no other symptom was noticed. The fever, which had returned with regularity each day at two o'clock, seemed to be of shorter duration, lasting only, on the 19th, until half-past three, and this, in conjunction with the fact that the peculiar chill and sweating stage, generally associated with malaria, were noticed, this diagnosis was provisionally made.

Diet becoming of importance, peptonized milk was advised, but this was rejected, the vomiting causing an increase in the intensity of the headache. This condition persisted, without any change or any improvement in the symptoms, until May 28, at which time examinations of the lungs, made daily, disclosed small patches of congestion over the right base. Cough became noticeable and quite irritating in character, but there was no expectoration. A cough mixture, consisting of—

Hydrocyanic acid, dilute, ℥i ;  
 Chloride of ammonium, gr. v ;  
 Syrup of marshmallow,  
 Syrup of white pine, aa fʒss,

was ordered to be taken every three hours. Under this treatment the cough, though still persisting, became less annoying, owing to the fact that expectoration became established. The sputum was whitish in color and thick in consistency, but showed no tubercle bacilli.

During that time daily examinations of the urine were made, the specific gravity varying between 1004 and 1009, odor normal, the color light, and the reaction markedly acid. Albumen was recognized by Heller's and the heat and nitric acid tests, showing a narrow ring, but there was no sugar. Microscopic examination

110000

showed some mucus, a few pus-cells, and at times several uric acid crystals, many large epithelial cells, but no casts nor cylindroids.

Examination of the blood for the malarial parasite proved negative, the red blood-corpuscles were markedly decreased, and the percentage of hæmoglobin reduced. Examination of the *fæces* showed no typhoid bacillus nor any *amœba coli*.

At this time, the diagnosis being absolutely unsettled, and consultation being requested, such was called for on the 28th of May. After the consulting physician had examined the patient he decided that the diagnosis laid between pernicious anæmia, tuberculosis, and typhoid fever. At this time the patient was in the following condition:

The headache still persistent, the fever at times as high as  $104^{\circ}$ , with a pulse of 132 and respiration of 26, would be observed towards evening, although occasionally the patient would show only a very slight rise in temperature. The lungs showed undoubted symptoms of catarrhal pneumonia. The bowels were normal in frequency, color, and consistency. The urine was passed in the usual quantities. No symptom except the headache was found which could be referred to typhoid fever. No symptom beyond the catarrhal condition of the lungs was noticed which would confirm the diagnosis of this disease. The diagnosis as a result of the consultation was, in view of the patient's past history, pernicious anæmia. The patient refused all food; even that of the blandest character would be immediately rejected; in fact, the very sight or thought of nourishment was sufficient to cause intense nausea and, at times, the vomiting of a glairy, viscid fluid. Once or twice, up to that day, the patient had a craving for unusual food, such as ham sandwiches or sour-kraut. Attempts to introduce alcoholic stimulants met with decided objection, as the girl positively refused to permit any alcohol to pass her lips. Requests of the family to permit the bringing of a trained nurse into the household met with a blank refusal, upon the plea that the patient would fight strenuously against the attendance of a stranger, and, recognizing the nervous state of the patient, this request was not insisted upon. Matters were continuing in that state from bad to worse, until, finally, on the 7th of June, Dr. Judson Daland was called in consultation. Both Dr. Daland and myself were forced to acknowledge that we were dealing with a case the diagnosis of which was very obscure. The patient had meanwhile

become greatly emaciated, owing to the failure of introducing a sufficient amount of food into the system, and I decided to insist upon the employment of a trained nurse. Dr. Daland examined the patient, and found the *status præsens* as follows: The lung shows unquestionable signs of catarrhal pneumonia, the cough small, expectoration yellowish white and thick, no blood, the abdomen soft, no tympanites, no spots of tenderness, no gurgling, the heart strong, acting excellently. Examination of the urine as usual; examination of the sputum showed no tubercle bacilli.

It was finally decided to treat the case as one of typhoid fever, and obtaining, two or three days later, a drop of blood, and treating this by Widall's method, the usual typhoid fever reaction was obtained. The treatment then advised was brandy one-half ounce every three hours, koumiss four ounces every four hours, and hypodermically strychnine one-twenty-fifth of a grain every four hours, and hyoscine one-one-hundredth every six hours. Per rectum, brandy three ounces, milk thirty ounces, and six whites of eggs in twenty-four hours.

Before beginning the nutritive enemas the colon was washed out with one quart of warm water containing two teaspoonfuls of salt, and the result was a dark, thin stool, consisting mostly of mucus. The temperature on the 9th at seven p.m. was 105° F., pulse 122, respiration 28. The patient was sponged with tepid water which was gradually cooled, and the temperature reduced in three hours to 104° F. This treatment was continued, and nutritive enemas and stimulants by the mouth were well retained. On the 10th, about eleven a.m., the patient became slightly delirious and restless, and the ice-cap was applied, improving the condition of the patient, who, however, again became delirious at seven the following morning. At this time tympanites became noticeable, and, supposing it to be due to an accumulation of gas, due to the nutritive enemata, colonic washing was again tried, resulting in the passage of a large amount of flatus, which in turn considerably reduced the tympanites. Gaseous eructations were then observed; the patient's lungs showed marked improvement. Until the 12th no new symptoms were observed, but on that day the first rose-spots were noticed on the abdomen, this being the thirty-fourth day of the disease. The patient from then on became delirious and very restless. It

became necessary to catheterize every five hours, as it was shown that the restlessness was due to fulness of the bladder.

The stools became now typically typhoid in character and appearance, foul-smelling, thin, greenish-yellow in color, with small particles of undigested food floating around. At this time the patient was taking three ounces of brandy by the mouth, and per rectum twenty-four ounces of milk, three ounces of brandy, and six whites of eggs, in twenty-four hours. Koumiss had to be discontinued, as it excited vomiting. Hyoscine was used at times, in combination with morphine one-fourth of a grain and atropine one-one-hundredth of a grain, to overcome restlessness. The physiological limit of strychnine having been reached, nitroglycerin one-one-hundredth of a grain every four hours was substituted. Calomel one-sixth and salol two grains were given every two hours. No improvement in the patient's condition was observed. The patient by the 14th of June was holding her own fairly well. On that day at four P.M. one stool was passed, mixed with a considerable quantity of clotted blood, showing the presence of internal hemorrhage. An injection of forty grains of tannic acid and five ounces of warm water was immediately given per rectum, followed by thorough stimulation. The patient became worse and sank very rapidly. Rest and quietness being so strongly indicated, opium suppositories, consisting of one-half grain of the extract, were given until the patient became slightly somnolent. Hypodermic injections of strychnine, nitroglycerin, whiskey, and aromatic spirits of ammonia were used to overcome the depression incidental to the internal bleeding, and as a result of this treatment the patient recovered wonderfully, until by the following morning she was considerably better.

During all this time attempts to keep down the temperature by cold sponges met with but poor results. About this time, nutritive enemata being out of the question owing to the condition of the bowels, an attempt was made to give nourishment by the mouth. This met with success, the patient taking from two to two and a half pints of milk in twenty-four hours and about four ounces of brandy. The milk was specially prepared by emulsifying it thoroughly with white of eggs and lime-water, and in that combination it was very well retained. On the 16th another hemorrhage was noticed, which was controlled as before.

During this time sleep was very much disturbed, the patient

being nearly the entire time in muttering delirium. The bowel movements now became brown and contained particles of fecal matter, but still possessed a very foul odor. The pulse was fairly strong, the heart was acting well, never relaxing its vigor.

On the 18th, at seven A.M., the nurse reported to me that the patient was "burrowing her head into the pillow," and on seeing the patient she was found to show symptoms of meningitis. A spinal ice-bag was applied, and as a result the patient became very quiet, sleeping for two and a half hours, but on awakening, the tremor was again noticed.

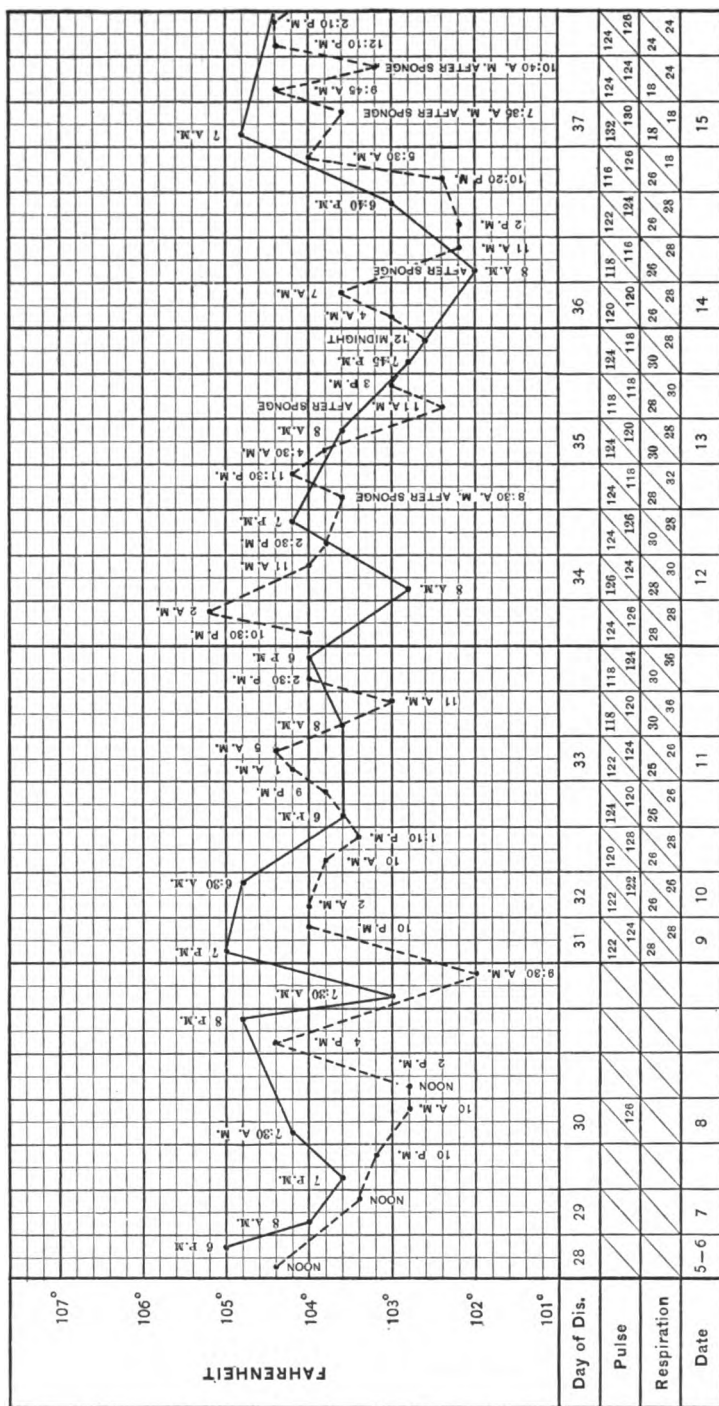
On the 19th the nurse reported "nausea following two alternate feedings, patient restless, opisthotonos and muttering delirium, stools and urine passed involuntarily, the temperature coming gradually down, the pulse and respiration more rapid." The pulse was markedly weaker and dicrotic. It was then recognized that the patient had only a very few more hours to live, although all the symptoms markedly improved by twelve o'clock the following morning.

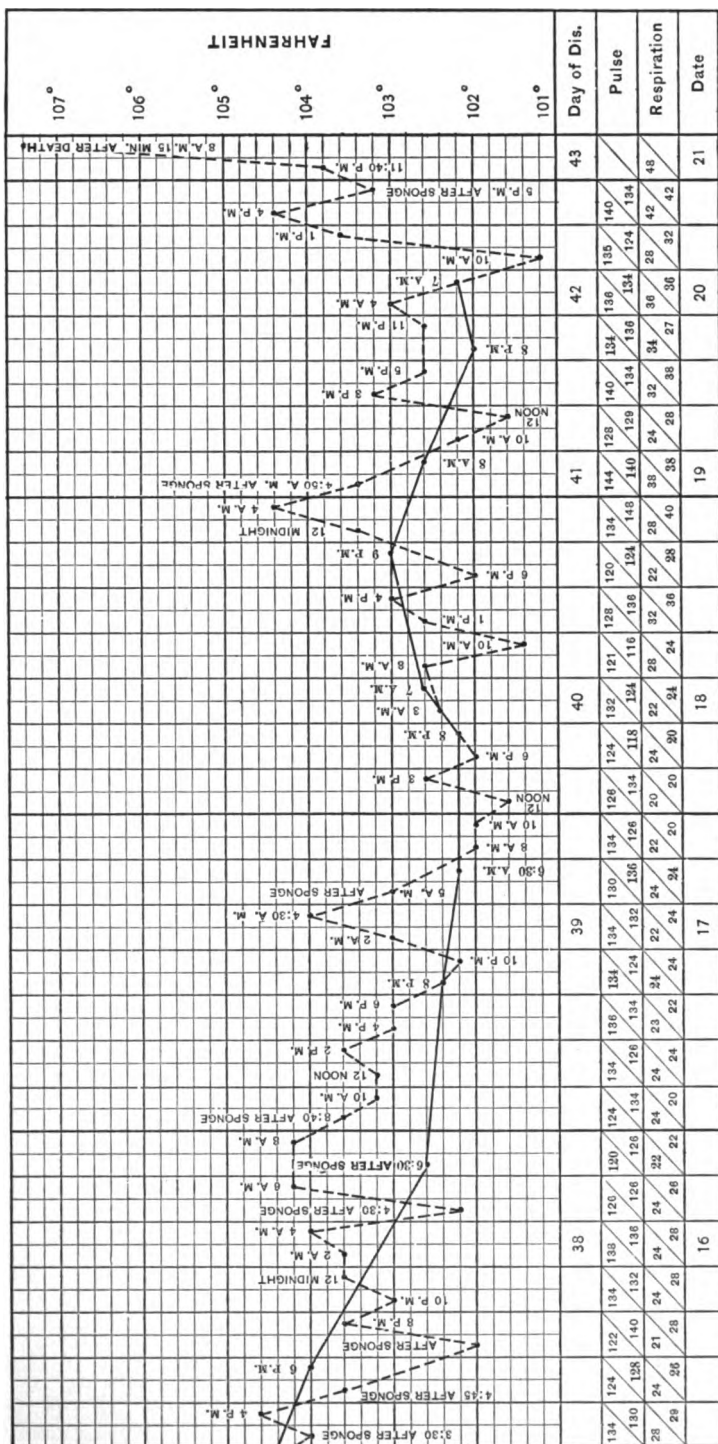
Throughout this illness, even in its worst stages, the heart acted with full strength and vigor, at no time abating. In fact, up to the very second of her death the heart was equally strong and full in its action. The patient died on the 21st at 7.45 A.M., the forty-third day of her illness, and passed away very quietly without the slightest struggle. Her respirations simply became weaker and weaker, until finally they became hardly noticeable, then disappeared. About five minutes after death the temperature which was taken was 107° F.

This case proved of exceeding great interest to both Dr. Daland and myself, in the fact that we had to deal here with a case which showed absolutely no symptoms for more than four weeks, showing only the presence of a slight congestion of the lung. In the fourth week symptoms appeared which usually are noticeable about the second week of typhoid fever, and from then on the typical typhoid fever condition.

The question, Did we have to deal here with a veiled case of typhoid fever at the very beginning, or simply with typhoid fever following catarrhal pneumonia? is still left undecided. We recognize that here was a greater temperature than normal; for forty-three days it was only once below 102° F., and the usual point







between 103° and 104° F. The heart never lost its power. It becomes a question whether or not the enforced starvation which the patient suffered previous to the introduction of the trained nurse did not in great measure help to bring about this condition which finally ended fatally. That a case of typhoid fever may be present in its fullest intensity without showing one single symptom usually observed in this disease must be recognized, as there is no question that the typhoid fever condition must have been present long before any symptom appeared.

It was agreed between us that the therapeutic measures—namely, the employment of remedies most generally used in typhoid fever—were out of the question. Owing to the very unsatisfactory condition of the stomach, we were forced to put the stomach at rest, introducing only the most important remedies per rectum or hypodermically. As soon as the stomach became retentive, it was employed solely for digestive purposes, and we were very careful not to introduce anything which might have removed the only way of introducing the very much needed food.

Taking the case as a whole, it was certainly very unsatisfactory, owing to the fact that I was forced to combat two prejudices,—namely, the one of having an educated assistant as a nurse, and the other the use of alcoholic stimulants.

To resume, I should think that this is one of the cases which show us the necessity of early interference in all conditions in which the symptoms are severe, the diagnosis obscure, and where it might prove more advisable for the physician to retire rather than permit a refusal of his demands for such an important factor as a nurse on the grounds of sentiment.

I feel to-day that if it had been possible to have the assistance of a person well versed in the taking care of the sick this fancied objection and rejection of alcohol and food would have been quickly overcome.

## CHLOROSIS: ITS COMPLICATIONS AND TREATMENT.

CLINICAL LECTURE DELIVERED AT THE MIDDLESEX HOSPITAL.

BY CECIL Y. BISS, M.A., M.D. (Cantab.), F.R.C.P.,

Physician to the Out-Patients' Department and Lecturer on Pharmacology and Therapeutics in the Middlesex Hospital Medical School, London, England.

---

GENTLEMEN,—In the choice of this subject and the preparation of the following observations I have been guided mainly by the consideration of utility. Chlorosis is so common a disease, and it will be so often met with in the practice of each one of you, that the examination of the points I hope to bring forward cannot fail to be of practical interest. As it has fallen to my lot to see a very large number of cases of it, the conclusions I submit to you are largely the outcome of practical experience.

When a disease is of common occurrence, and but rarely fatal, there is always a tendency to regard it as of slight importance. Chlorosis, however, though it usually tends to recovery, is a malady which sometimes induces serious consequences, and is occasionally fatal. Recurring attacks of it—and it always tends to recur—often induce so much debility as to facilitate the invasion of the gravest diseases, or to induce a chronic condition of ill health. Beyond this, however, chlorosis has its own dangers, such as cardiac dilatation, thrombosis, gastric ulcer, etc.; and as it is never in our power to determine, during the early history of a case, whether one or another of these complications may not supervene, we ought always to be alive to the necessity of exercising caution, and to give a somewhat guarded prognosis. It is the conviction that there is a somewhat general disposition to regard the dangers of chlorosis too lightly that forms part of my motive in bringing the subject forward.

Most of us are, I suppose, in the habit of loosely calling chlorosis anæmia,—and anæmia it is in the broad physiological sense of being a condition in which there exists a deficiency in the blood,—

but there is no essential similarity between true chlorosis and other forms of anæmia, such as anæmia from hemorrhage, from malaria, from tuberculosis, or the so-called pernicious anæmia. Sometimes it happens that this distinction is not observed,—in other words, that the diagnosis is not correctly made,—and then treatment is apt to fail. It is not my intention, however, to discuss the differential diagnosis of chlorosis, except as to one condition from which it is sometimes extremely difficult to discriminate it; I mean the anæmia which often ushers in, and is, indeed, itself one of the commonest symptoms of, early tubercular disease. Here is a brief note of a case illustrating this:

Miss C., aged eighteen, was brought to me by her mother in November, 1890. She was said to be suffering from a cold and sore throat. Her family history was good, except that one maternal uncle had died of consumption. She was decidedly anæmic, and had a faint systolic murmur at the apex of the heart, judged to be hæmic in origin, with some catarrh of the pharynx. A month later she had a little pain in the right lower axilla, where a slight friction sound was audible. Her health did not improve much under treatment, and when, a year later, she was again brought to me, suffering from slight cough and loss of strength, moist sounds were detected at the right apex, and the diagnosis of tuberculosis was obvious; of which disease she died about two years later. In this case there can be little doubt that the original anæmia was not chlorosis, but an early symptom of tuberculosis, to which cause the attack of dry pleurisy was also due.

Trousseau<sup>1</sup> dwells upon this variety of anæmia, which he describes as a "false chlorosis," and deprecates its treatment with iron, saying that, although it may be apparently improved, the development of tubercle will be hastened by such treatment. His words are, "I would wish to tell you that you will avoid distressing occurrences in your practice by allowing patients predisposed to tuberculous disease to retain maladies from which it may seem opportune to deliver them." He does not make it clear that these conditions of true and false chlorosis can be distinguished except by the results of treatment by iron, which will cure chlorosis, but, as he believes, probably develop false chlorosis into tuberculosis. Perhaps, however, it will not be too much to say that the views of this great physician would

---

<sup>1</sup> Clinical Lectures, Sydenham Society edition, vol. v., p. 95.

probably have been modified if he had known, as we now do, that tuberculosis is a disease due to infection by the tubercle bacillus; and it seems probable that the cases he describes as "false chlorosis" are really cases in which tuberculosis was already existent, though latent, and in which it would have become developed sooner or later, whether treatment by iron had been adopted or not. But it may also be the case that we have here an obscure failure of general nutrition, one of the features of which is a great impairment of the blood-making functions of the body; and that it is this failure of nutrition which gives its opportunity, so to speak, to the invasion of the tubercle bacillus. Now, chlorosis by producing debility may induce tuberculosis, yet in other cases an anæmia difficult to distinguish from that of chlorosis may itself be an evidence, as in the case quoted, that the tubercular process has begun.

It would be beside my purpose to discuss the etiology of the disease, but I venture to remark upon one point because of its important bearing upon the question of treatment. The causation of chlorosis is still surrounded by a measure of obscurity. Sir Andrew Clark's theory of constipation was, in all probability, a contribution in the right direction; although, in common with many others, I cannot think that it is a complete explanation. The latest views point towards chlorosis being the result of an impoverished nutrition of the blood resulting from the decomposition within the intestine of those organic compounds comprised in food which contain iron in an assimilable form; which decomposition is produced by the noxious action of certain inorganic agents, probably alkaline sulphides, whose presence and effects may be due to inaction of the bowel. If this view be correct it explains much: it throws light, in the first place, upon the undoubtedly beneficial action of aperients; and it goes far to explain the interesting fact, of which there is no other explanation, that large and even enormous doses of iron do so much more good than small doses; and also why certain preparations of iron, such as the carbonate, are so much more beneficial than the others. There is much reason to suppose that chlorosis is a disease due to causes which interfere with the proper genesis of the hæmoglobin, and, to some extent, of the red blood-corpuscles.

We come now to consider the complications of this disease,—a part of the subject which does not always receive the consideration it deserves, seeing that these complications are both numerous and serious. They are,—

## NERVOUS DEBILITY.

All nutrition must depend eventually upon the blood: poor blood must, therefore, induce an imperfect production of nerve-force. This is just what is found in chlorosis: its subjects suffer from lassitude, muscular weakness, headaches, and many other symptoms, all of which may be reasonably attributed to nerve-prostration. With the amelioration of the disease these symptoms improve, but it often happens that the general health remains long impaired, and the general constitutional vitality much lowered; and as this occurs most frequently in the early years of adult life, and that principally in the female sex, who are specially prone to neuroses, it is obvious that the seeds of many troubles may be thus sown, and evil effects produced which can never be wholly rectified. Thus, as Fagge points out, chlorosis is often the starting point of phthisis, and hysteria, chorea, gastric ulcer, and exophthalmic goitre are particularly apt to occur in those who have been affected by it. These are prominent examples of the fact just noticed, but it is extremely probable that many slighter forms of disorder such as migraine, neuralgia, feeble digestion, neurasthenia, and other disturbances of nerve-health are the direct, and it may be the lasting, results of recurrent attacks of chlorosis.

## CARDIAC DILATATION.

This is a common and often a grave complication. To it is mainly due the breathlessness on slight exertion so often noticed; yet not wholly so, for the impaired power of the red corpuscles to absorb oxygen, owing to their poverty of hæmoglobin, must also interfere greatly with the respiratory function. This dilatation is no doubt the result of the enfeeblement of the cardiac walls resulting from malnutrition, being, in fact, a part of the general muscular weakness; and the sluggishness of the circulation thus produced is an important factor in the production of thrombosis, to which these patients are sometimes liable. Cardiac dilatation in chlorosis is sometimes productive of grave consequences. It is true that it is usually recoverable from, but when neglected, or in cases where it recurs again and again, it is no doubt capable of inducing permanent structural changes in the heart itself.

## THROMBOSIS.

This is an occasional, though perhaps not very frequent complication; yet it may occur in any case, and involve the gravest dan-

gers. The formation of thrombi in the veins is always to be dreaded when the blood is poor and the circulation feeble, as, for example, in the later stages of consumption and cancer. It may occur in chlorosis with fatal results. I can recall the case of a young woman admitted into the hospital in a semi-comatose condition, with a raised temperature, but no physical signs except those indicative of chlorosis. The coma deepened and she died in a day or two. Clots were found in all the cerebral sinuses, and to this cause solely her death was due.

#### ULCERATION OF THE STOMACH.

All observers admit that gastric ulcer frequently occurs in chlorotic subjects, so often, indeed, as to suggest a definite, though as yet obscure relation between these conditions; but I have a strong impression that ulcers form much more frequently than is usually thought, for in many patients who have been thought at first to be suffering merely from dyspepsia, or gastralgia, undoubted symptoms of ulceration subsequently supervene. The method of production of these ulcers is not fully understood, but they are commonly referred to two causes,—first, lowered vitality of the tissues; and second, the formation of a thrombus in one of the gastric vessels, inducing necrosis in the neighboring area of mucous membrane, the blood-supply of which has been interfered with; the necrosed tissue is gradually eroded by the digestive action of the gastric juice, and becomes hollowed out into an ulcer. The clinical importance of this point is obvious, for when ulceration is suspected the patient can be put under appropriate treatment and the evil effects of the condition minimized. If, however, the probability of ulcer be overlooked and the case regarded as one of merely functional digestive disturbance, the treatment is likely to be ineffective, and the formation of a perforating or chronic ulcer may follow.

#### GASTRALGIA.

Apart, however, from ulceration of the stomach, it is exceedingly probable that gastralgia—by which I mean gastric neuralgia without any organic lesion—may occur as a very distressing complication of chlorosis. It may be difficult, and sometimes even impossible, to distinguish such cases from those of ulceration, but, nevertheless, they do occur. Whenever there is any doubt it is safer to lean to the view of ulcer, and to treat the case accordingly. Ten



years ago I saw, with her medical attendant, a young lady, aged eighteen, who was suffering from palpitation and severe pain over the stomach, but without epigastric tenderness. She was intensely anæmic, and had been treated with many drugs without any benefit. I suggested that she should be put to bed, and kept at perfect rest for a fortnight, taking in the first place only a milk diet and some pills of carbonate of iron. Her relief was immediate and complete. The pain, which had been severe even to the point of agony, never recurred, and she made a rapid recovery. In this case it is probable that gastralgia existed apart from ulcer: I judge this from two considerations,—first, that there was no vomiting; and second, that the pain immediately subsided and never recurred after she was kept at perfect rest. This is not an isolated case; probably all have seen others resembling it.

#### DILATATION OF THE STOMACH.

In all conditions of general debility, and, therefore, in chlorosis, there is a tendency to dilatation of the gastric walls, and in this way a greater or less degree of dilatation may become a complication in chlorosis. I need hardly point out that cicatricial contraction resulting from old ulceration in the neighborhood of the pylorus may also produce dilatation from obstruction. Both these conditions have to be borne in mind.

I will arrange what I have to say regarding the treatment of chlorosis under the following heads:

#### REST.

I have no hesitation in placing first in order of importance, among therapeutical measures, the curative agency of rest. Chlorosis is a disease of debility, and its features are those of debility, whether of the whole system or of particular organs; it is reasonable, therefore, to believe that the first condition of improvement is to secure the fullest measure of mental and bodily rest which is possible. A quiet bedroom and a warm bed are everything to a patient suffering severely from chlorosis. Many cases which are intractable while the patient is up and about begin to improve directly she is put to bed and all strain removed. Generally speaking, I believe that rest is required in all cases of chlorosis,—certainly in those presenting the complications of cardiac dilatation or gastric ulcer,—and that in all but the slightest cases complete rest for a short period is the

best way to inaugurate the treatment. It is a good rule to enjoin perfect rest in bed with as little excitement as possible for the first ten days or fortnight; for the second fortnight, to direct that the first half of every day be spent in bed, and the second upon a couch; for the third fortnight, that the patient should rise after breakfast, but take only moderate exercise, driving out or sitting in the open air in preference to taking walks. This may frequently be impracticable, but I believe it is wise to get as near it as possible in practice, for sometimes the debility and depression induced by the disease are so great that the organism appears incapable of responding to treatment until the full therapeutical value of rest has been realized. Let me quote an illustrative case:

Miss H., aged twenty-six, residing in Bedfordshire, was brought to me in July, 1891, with a history of having been "pale and ill" for six years. She had shortness of breath, palpitation, indigestion, constipation, amenorrhœa, morning retching, and swelling of the legs and feet. She had been treated by more than one practitioner, and with various medicines, but had not improved, and had become so weak that she could scarcely walk. The case appeared to be one of pure chlorosis. She was sent home and kept in bed for several weeks, care being taken to secure her complete rest, regular action of the bowels, and liberal feeding up to the level of her digestive powers. She took twenty grains of carbonate of iron in a pill thrice daily. Her improvement, though slow, was steady, and in three months she had completely recovered. Before the treatment was commenced she was regarded by her friends as a dying woman.

It is hardly needful to point out that the cases in which cardiac dilatation exists especially require rest, and that in those of gastric ulceration, gastric rest, as well as general rest, is necessary. In the worst cases, complete rest of the stomach for a few days, during which rectal feeding should be carried on, is advisable, and in the slighter cases a fluid diet administered in small quantities at appropriate intervals; but I do not think that the presence of a gastric ulcer is necessarily a hinderance to the administration of iron if this be given as the carbonate, which is a mild preparation, and in the form of a pill; indeed, it is advisable to commence the treatment by iron as soon as possible, so that by the improvement of the blood healing may more readily proceed. I have never yet seen a case in

which iron produced gastric disturbance if given under these conditions.

#### APERIENTS.

Aperients are needed in almost all cases; and when the patient is kept at perfect rest they are more than ever necessary. It is well to commence the treatment with a mercurial pill and a saline draught, after which it is sufficient to keep the bowels freely open by daily doses of cascara sagrada, or mild pills containing aloes and belladonna.

#### FOOD.

A chlorotic patient should be abundantly fed, yet not at the beginning of the treatment, nor where there is any suspicion of gastric ulcer. Treatment is generally best commenced by nourishment in the fluid form, such as eggs beaten up in milk, custard, etc., to be followed by milk puddings, scraped raw beef in small quantities, then pounded meat or chicken, and then fish and meat. As soon as it is certain that the digestion will bear it, it is well to feed liberally, and in some cases massage may be usefully employed to facilitate digestion and assimilation.

#### IRON.

I believe that iron is still the best specific for this disease, and that none of the other drugs which have been recommended are to be compared to it in usefulness. Experience has, however, convinced me that two conditions should be observed in its administration: first, that only one of the milder preparations be given, and of these none is better than the freshly prepared carbonate; and secondly, that this should be given in the form of pills, or tabloids, which are not likely to be disintegrated until they reach the intestine. The *pilula ferri* of the Addendum to the British Pharmacopœia is very useful. Liquid preparations of iron, and especially those of the stronger forms, such as the perchloride, should be avoided, because they tend, owing to their strong astringency, to irritate the stomach; in cases of ulcer they would of course be absolutely inadmissible, while pills or tabloids containing the carbonate may be given in practically all cases. An essential point is that the iron should be administered in sufficiently large doses. My own rule is to give three and frequently four of the *pilulæ ferri* just mentioned thrice daily after food; and I believe that the apparent failure of iron in some cases, or its very gradual and partial success, is due to the fact that a suffi-

cient quantity is not administered. Of course it is not suggested that this large quantity of iron can be absorbed; probably none of it is absorbed; but it may act usefully in a different way. The recent researches of Bunge go to prove that iron in inorganic forms is not assimilable at all, and that its usefulness in chlorosis is due to the power it possesses of neutralizing alkaline sulphides in the bowel, which, if unneutralized, would decompose those organic compounds in the food which contain iron in an assimilable form, and thus prevent the system being robbed of the iron which it needs for the formation of hæmoglobin. If this view be true, it is easy to understand that larger doses of iron may be required than have hitherto been thought necessary; but whether this be the true explanation or not, the fact that a case of chlorosis recovers more rapidly on large doses of iron than on small ones is, I think, unquestionable.

## THE TREATMENT OF CHLOROSIS.

CLINICAL LECTURE DELIVERED AT ST. ANTHONY'S HOSPITAL.

BY PROFESSOR HAYEM,

Clinical Professor of Medicine in the Paris Faculty of Medicine, and Visiting Physician to St. Anthony's Hospital, Paris, France.

---

GENTLEMEN,—We have recently had in our wards a certain number of cases of chlorosis. Among the patients who have already left us, one, who returned to her home before she was cured, would have enabled me to show you the tardy variety of this disorder; those who still remain are examples of the chlorosis of young girls of the chloro-dyspeptic type, as they are all manifestly dyspeptic, which is the rule in such cases. Two of our patients are of this variety, the most frequent, one of their neighbors is affected with chloro-tuberculosis, while still another will give you an example of serious anæmia produced by gastric hemorrhage,—the chlorosis in her case being complicated by gastric ulcer.

I shall take advantage of the presence of these patients to lecture on the treatment I have formulated for these cases, leaving on one side the historical details of the question, as well as all discussion of the many different treatments that have been proposed by other writers of late years.

The treatment of chlorosis should comprise three classes of means, each of which fills to an equal degree an important indication; the treatment, therefore, must be used in its entirety to insure success:

- (A) Rest.
- (B) Regimen and gastric treatment.
- (C) Iron in a suitable form.

(A) *Rest*.—For a long period it was customary to advise physical exercises,—walking, gymnastics,—which were supposed to be excellent means for stimulating the general circulation, for awakening the appetite, and giving strength. I had noticed, however, that

hospital patients, who were kept in bed, got well more rapidly than private patients. In addition to this, by following in these patients the quantity of pigment of hæmatic origin eliminated, I saw that it was more abundant after a day of exercise than after a day of rest. These considerations appeared to me to warrant my keeping in bed my private patients as well as those in my hospital wards, and I have been so satisfied with this method that I now consider it a requisite condition of success, and especially of rapid success.

Confinement to bed, which I have advised since 1881, is indispensable in bad cases, very favorable in moderate cases, and useful even in light ones.

It accomplishes several purposes which I must point out to you:

1. It counteracts too rapid destruction of globules, and I have already told you that chlorotic anæmia is due,—at any rate, in part,—to exaggerated destruction of red globules.

At the confirmed stage of the disorder the urine is only slightly colored, of a pale and special green tint, provided the patients remain in bed. It is, on the contrary, colored, and gives the reaction of urobilin and urohæmatin when the patients are up and take any exercise, no matter how slight. Is this not a proof that work favors globular destruction, just as rest moderates it? Consequently, when we give iron to certain patients with chlorosis, without keeping them in bed, they lose in one way what they gain in another.

2. The majority of these patients, particularly the cases of dyspeptic chlorosis, suffer from neurasthenia as well; they are out of sorts, more fatigued on awakening in the morning than when they went to bed, and irritable to excess. Rest in bed is an efficacious remedy for this neurasthenia, lessens their irritability and relieves fatigue, restores nervous equilibrium and regulates nutrition, and produces sleep or makes it more refreshing.

3. Finally, rest has the advantage of doing away with corsets. Without subscribing to the exaggerated ideas of Meinert, who looks on chlorosis as one of the forms of the corset-disease, there is no question but that this article of apparel conduces strongly to dilatation of the stomach. Rest in bed enables the stomach to come back to its proper dimensions, and as soon as your patients begin to go about again you should get them to adopt an elastic corset.

No fixed rule can be enunciated as to how long your patients must be kept in bed, nor should all these patients be put in bed for

the same length of time. The length of time should depend on the degree of the anæmia, on the intensity of the neurasthenic condition, and, I may also say, on the dyspepsia.

In light or moderate cases, which are those usually observed, four or five weeks is sufficient time. Serious cases with great anæmia and asthenia require six to eight weeks, or even more. It is also advisable to bring the patients by progressive steps from complete rest back to their ordinary life: first let them get up for their meals only, then let them drive out, or lie out-of-doors on a lounge if the weather permits. You can then let them move around, fixing the time for them,—two hours at first, then two hours and a half, increasing steadily.

Absolute rest, which I have recommended for fifteen years now, was at first adopted by my pupils, but is now used by many physicians in Germany and England, as well as in France. At one of the last German medical congresses it was approved of by Nothnagel, Ziemssen, Edlefsen, etc.

(B) *Regimen and Gastric Treatment.*—The second portion of the treatment is not less important than the first. Chloro-dyspepsia,—that is to say, chlorosis preceded and complicated by a gastric condition,—represents the commonest form of chlorosis. I have laid sufficient stress on this point in my preceding clinics to make any further explanations unnecessary.

The causes of this gastropathy are the same as with young girls who are not chlorotic. Heredity, direct or indirect, the latter represented by disorder of the nutrition in the parents, diseases of childhood that were followed by gastric lesions, defective alimentary hygiene, finally, the use of corsets, which very frequently cause dilatation, are among the principal causes of the gastric disorder.

You understand that the dyspepsia creates a favorable situation for the occurrence of chlorosis, but does not really beget it; that it is not directly proportionate to the intensity of the anæmia; that it survives the recovery from the chlorosis, and that it is often made worse by ill-advised treatment.

Clinically, therefore, you will find in chlorotic patients, particularly when they have followed no treatment, a hyperpeptic type of gastritis with a greater or less amount of dilatation; in less frequent cases a more advanced form of mixed gastritis with glandular atrophy, or a hypopeptic type caused by the chronicity of the

gastric lesion, the peculiar origin of this lesion (such as infections, which facilitate the degeneration of the mucous membrane); oftener still, medicinal irritation, which is capable of giving rise to new lesions in addition to those of the original gastropathy.

You will now realize the necessity of making the diagnosis of the gastric condition before beginning any treatment; this is a rule from which you should on no account deviate.

There are, furthermore, several very good reasons why you should inquire into the digestive functions:

1. The improvement of the digestive process facilitates the reconstitution of the blood and of the entire system.

2. Iron is always a remedy difficult to digest, with a tendency to make the dyspepsia worse. It is therefore advisable to prepare the stomach by a suitable treatment to facilitate its absorption. Otherwise your chlorotic patients may remain dyspeptic although they recover from their chlorosis, and even then this recovery is only relative.

3. The gastropathy creates a strong predisposition to relapses; recovery is, therefore, more complete and lasting when the dyspeptic condition is cared for as well as the anæmia.

The physicians who spoke at the Munich Congress do not seem to have laid much stress on the regimen. Some of them (Ziemssen, Baumler) were of opinion that iron can be administered in spite of the dyspeptic condition. This is quite true, particularly when a suitable preparation of iron is chosen; but I do not agree with von Ziemssen's counsel when he says that in the presence of gastric atony, or of symptoms of gastric catarrh, iron must be given at once, without wasting any time in treating the stomach.

Experience has shown me that the treatment of the gastropathy, —at any rate, by diet,—so far from being time lost, is a distinct gain, both for the present and for the future.

Practically the cases can be divided into two groups as regards the gastric condition:

The first group, the most numerous, comprises the cases of moderate hyperpepsia without serious dyspeptic troubles, with slight or moderate dilatation, with or without mechanical hinderance by tight lacing.

After a few days of rest and diet you can administer iron, but



the diet must be a strict one, and the one I have used for a long time now is quite different from that generally advised in such cases.

At first, milk, milky soups, raw meat; two or three weeks later, soft eggs, fish with white meat, green vegetables, and stewed fruit. Bread should be allowed only at the end of four or five weeks. In some cases, when there is gastric pain, you will do well to advise the cold pack for the epigastrium,—at any rate, for the night.

The second group comprises about twenty per cent. of these cases. The gastropathic condition requires more special attention here, and we must consider two categories of patients.

To the first belongs parenchymatous gastritis with great dilatation; the regimen must be stricter and the number of meals more limited,—three or four a day, composed of milk and raw meat. Massage of the abdomen—that is, of the stomach and intestine—is useful, particularly when the dilatation is partly due to mechanical compression by corsets. In exceptional cases washing of the stomach is advisable, when there is fermentation with great acidity. This treatment should be maintained for two or three weeks before the specific treatment is commenced, and the latter should coincide with the administration of a more copious and varied diet.

In hypopeptic patients, with mixed, atrophic gastritis without complications, who form our second category, a less severe diet can be rapidly permitted and iron given at an early date; but a certain amount of hydrochloric acid should be given after the meal at which the iron is taken.

I am in the habit of prescribing this acid in the form of a one-per-cent. solution, of which I give a tablespoonful in a quarter of a glass of sweetened water half an hour after eating.

(C) *Iron in a Suitable Form.*—When I first began my researches concerning the treatment of chlorosis, in 1876, many physicians questioned the efficacy of ferruginous preparations. Yet their favorable action is not open to doubt, as I have proved in various publications.

My experiments with Regnault, in 1877, showed that a ferruginous preparation that cannot be assimilated by the organism (ferrocyanide of potash) is of no avail in restoring the quality of the blood. Iron combined with an organic radical (cyanogen) is no doubt readily assimilated, but it passes through the system without leaving any trace and without contributing to hæmatic renovation.

Trousseau claims to have obtained recoveries with the use of the peroxide of manganese which he had failed to effect with ferruginous preparations. Yet my researches on the action of protochloride of manganese and of arsenic have shown that no other means succeeds as well as iron, and that manganese in particular will not displace that remedy.

It remained to be shown whether chlorotic patients cannot obtain the iron they need from their food, and whether the iron treatment does not act with them, as some writers believe, by stimulating appetite and digestive power.

I tried in succession inhalations of oxygen, which increase appetite and facilitate digestion, and hydrotherapeutics, which also give good results, and in this way was able to get chlorotic patients to absorb a large amount of food, substantial and rich in iron.

When the anæmia is only slight or moderate noticeable improvement is effected; appetite is awakened, strength returns, the skin becomes tinged, and the number of globules increases; but in a short while (two to three weeks) the increase comes to a standstill, the newly formed globules remain imperfectly developed, and the characteristic lesion of the blood persists, even when the treatment is carried on with perseverance for several months longer.

In some cases, even, where the anæmia is very marked, hydrotherapeutics fatigue the patient, prevent sleep, and make the situation worse.

After having noted these facts in chlorotic patients subjected to these different treatments, I then prescribed iron, and in all cases promptly obtained, if not permanent, at least temporary, recovery.

By comparing these cases with those in which I advised the iron treatment from the outset and obtained rapid improvement in the quality of the blood, I concluded that iron, thanks to the part it takes in the composition of the blood-globules, has a specific action which no other remedy or treatment can replace. This action shows itself in blood in which the globules no longer have a normal evolution by a more or less rapid return to the physiological type.

Iron is the specific remedy for chlorosis; it assists in globular renovation, and, after checking the process of deglobulization, it enables the red corpuscles to reach their full development.

This opinion is entirely different from that of Bunge, of Bâle, who, relying on experimental researches, has for many years main-

tained that inorganic preparations of iron are not absorbed by the digestive tube to any appreciable amount; that our tissues derive the iron they need from the organic compounds contained in the food, in meat particularly; and that the preparations of inorganic compounds so commonly used in practice and given to chlorotic patients are of very questionable use.

It should be added that these opinions, presented lately to the Munich Congress, were not received with favor. All the authorities who spoke on the question admitted the practical value of iron in chlorosis, so that the matter is definitely decided.

You will have to use some preparation of iron, as the action of ferruginous mineral waters is inappreciable. The choice of a preparation of iron is not an indifferent matter. I have used for a long period, now, the oxalate of the protoxide of iron, but you can prescribe almost any protosalt that will be readily transformed in the digestive tube.

I was surprised to see the majority of German practitioners prefer Blaud's pills (carbonate of iron), and recommend a long treatment with large doses. For myself, I prefer to give small doses, and for as short a time as possible. One of the advantages of the protoxalate is that you do not need to exceed 0.15 to 0.20 gramme (2 to 3 grains), at the most, twice a day. Under the action of this salt, the other rules of the treatment being properly carried out, the patients gain color almost while you are watching them, so that the remedy does not have to be administered long. I usually begin with 0.10 gramme (1.5 grains) at the beginning of or during the two meals; in eight to ten days I increase to 0.15 gramme or 2 grains. In some cases I go to 0.20 gramme, but never beyond; and this I do for one month, when I interrupt the treatment, resuming it again later if necessary.

Finally, you will complete and render permanent the recovery of your chlorotic patients by advising hydrotherapeutics, aërotherapy, high altitudes in some cases,—that is to say, by adding to the primordial prescriptions of which I have been speaking the treatment by physical agents.

This whole treatment is unquestionably one of the most satisfactory among those that do credit to our profession, and I am very glad to see that it is being adopted by my confrères, both at home and abroad.

## THE TREATMENT OF PUERPERAL SEPSIS.

CLINICAL LECTURE DELIVERED AT THE KENTUCKY SCHOOL OF MEDICINE.

BY WILLIAM H. WATHEN, M.D., LL.D.,

Professor of Obstetrics, Abdominal Surgery, and Gynæcology in the Kentucky School of Medicine; Fellow of the American Gynæcological Society and of the Southern Surgical and Gynæcological Society; Gynæcologist to the Kentucky School of Medicine Hospital and the Louisville City Hospital, etc., Louisville, Kentucky.

---

### LECTURE III.

GENTLEMEN,—Having considered in the two preceding lectures the etiology and pathology of puerperal septic infection based upon the most recent investigations, we understand that the medical profession now recognize that this disease in its manifold manifestations is of bacterial origin,—is a germ disease. This being true, the treatment must in a degree be germicidal, but absolute cleanliness is of paramount importance in the prevention of all forms of septic infection. Where the surgeon or obstetrician believes that he can prevent septic infection by the use of germicides, disregarding cleanliness, he will be disappointed, and his results will be practically no better than were the results of those physicians who lived before we knew anything about the action of germs in the causation of disease. The action of germs in producing puerperal septic infection was probably not understood until about twenty years ago, and not generally understood until within the last few years. It is true that Semmelweis, of Vienna, in 1847, called attention to his belief that puerperal or childbed fever was a septic disease, but he did not convince the medical profession that his views were correct, and they were not accepted or recognized. He finally died in a mad-house, and it was a generation after he promulgated his views on this subject before the medical profession began to believe that the disease was septic, and the obstetrician developed this subject more than the surgeon.

In 1886, Lister, basing his views upon the observations of Pasteur, taught the germ theory of disease, but it was not applied in any sense to obstetrics until 1870, when Stadfelt, of Denmark, and Bischoff, of Switzerland, separately recommended the use of carbolic acid in obstetrical practice, but their ideas of the nature of the disease and of the action of the antiseptic in its prevention and cure were so crude and seemed so impracticable that the profession did not adopt them; but in 1881 the great French obstetrician Tarnier recommended bichloride of mercury, which, in 1883, after the experiments of Koch, was generally adopted in obstetrical practice, and has probably in some cases been used too extensively and may have caused harm. But if we will study the morbidity and the mortality in the lying-in hospitals of the old world and of this country before and since the use of bichloride of mercury, we will find that the mortality from septic infection has been almost entirely eliminated. Before that time the mortality from sepsis in these lying-in hospitals was so great that it was cruel for a woman to be delivered in one of them. Now, the mortality is practically *nil*, and these hospitals are the safest places that we have in which to deliver women. In the New York Maternity Hospital we find that the mortality has been reduced to about one per cent., and for three consecutive years, the number of patients running up into the thousands, there was not one death from sepsis. This has all come about since the general use of and belief in the efficacy of bichloride of mercury as a preventive and curative agent in puerperal sepsis; but we must not believe that it is due entirely to the use of bichloride of mercury or to any other form of antiseptic, because if we do and neglect other means, we will find that many of our patients will become infected; but this remedy is useful and has its place, though the most favorable results are mainly due to the absolute cleanliness that the obstetrician and the surgeon have adopted since the introduction and use of antiseptics. So, then, we must depend upon antisepsis and surgical cleanliness; and if we have to dispense with one, let it be the former, because while antiseptics properly applied are of great value in obstetrics and in surgery, asepsis or surgical cleanliness is indispensable. We may obtain nearly as good results from the use of surgical cleanliness without the use of germicides as we will by the employment of these remedies, provided we are certain that we have availed ourselves of

every means to cause everything about the patient and the physician, etc., to be aseptic. If we do not give the matter the most careful attention, we will not preserve perfect cleanliness, and will not then prevent infection by the use of antiseptics; the same law applies to obstetrical work as applies to surgical work, and those men who get the best results are those who are the most cleanly; and many men who recommend the use of germicides have had results from septic infection, because they do not appreciate the value of aseptic precautions. They do not wash their hands, their instruments, or the person upon whom the operation is to be performed, or the woman who is to be delivered.

So the treatment in these cases must be preventive and curative; it must be local and general; medical and surgical; and just when we are to depend entirely upon one means, and when we are to depend upon another, cannot now always be positively determined. When we are to apply surgical treatment is often a difficult question to decide, and much injury has doubtless been done because of our imperfect knowledge of the subject, operating sometimes when surgical treatment is not indicated, neglecting to operate again when surgical treatment offers the only means of relief.

Now, as to the preventive treatment. We can accomplish more good in puerperal sepsis by the preventive than by the curative treatment, for there are certain forms of puerperal septic infection that are beyond means of control, and in which nothing in medicine or surgery is of any benefit; but we are able to almost entirely eliminate septic infection in obstetrical cases, as has been done for years in the large lying-in hospitals. How are we to do this? First, by instructing our patients to be thoroughly clean; to avoid association or contact with all conditions that are septic, or that may convey septic matter to the pregnant woman; and this should be observed especially just about the time the woman is expected to be delivered. She should not associate with any person or go in the presence of any person suffering with infectious diseases, such as scarlet fever, diphtheria, erysipelas, etc., and she should, before she goes into labor, thoroughly bathe herself and put on clean clothing; the attending physician should be certain that he has no septic matter upon his person, that he has not recently been treating septic or contagious cases, that his hands are made clean by the use of soap, water, and antiseptics, and that the woman's vulva and parts in the vicinity of

the vulva are also made clean by washing and by the use of antiseptics; that everything that comes in contact with the vulva and vagina during and after labor is also free from septic matter. We may be called to a woman in labor, and if she has not been otherwise advised, we may find lying under her an old bed-quilt or comforter that has been thrown aside as unfit for anything else; it is used upon this occasion because when it is soiled, being of no value, it may be thrown away. This in itself is enough to infect the woman. Everything of this sort, and everything about the bed and room, must be free of any such conditions as we have in this dirty clothing, and the woman must be covered with a clean boiled sheet, and have one under her also; if anything else is put under the patient it must be covered with a clean sheet. If we use instruments they must be sterilized, and we must carefully observe all the laws of asepsis and antiseptics that are required in any kind of surgical work.

The foregoing remarks apply to private practice. Of course, in hospital practice we may accomplish these results much better, because these patients are then entirely under our care, and we can subject them to any form of preparatory treatment that the hospital has adopted. By observing these means we will then have very few cases of septic infection, and will have few cases of infection even if we do not use bichloride of mercury or other germicide; however, there is no impropriety in using upon our hands or instruments, or upon the vulva, a germicidal solution when we have done all we can by washing with soap and water.

This is enough about the preventive treatment, and if we will follow it in all its details we will not have to treat many cases of septic infection; but we may in the practice of some one else be called upon to treat puerperal sepsis, and we must know how to treat it. The treatment depends upon the conditions with which we have to deal. In one instance we have an infection confined to the uterus and the vagina, with an under layer of granulation tissue and of leucocytes battling against invasion of the infection and protecting the system from this deadly poison. In another instance, there is a very thin layer of granulation tissue, and possibly not enough leucocytes to prevent further invasion of germs into the system. There may be no protecting layer of granulation tissue, and the leucocytes may have been overpowered, and the poison may have gained en-

trance through the lymphatics or the veins into structures around the uterus, or possibly into the entire peritoneum, or into the system at large. We have to take these cases as we find them, and we have to apply our treatment to the conditions that exist. If we have a putrid infection, with a foul odor, unless we can by examination with the microscope find the streptococcus or some of the pus-forming micro-organisms, we may infer that the disease has not gone beyond the endometrium, unless we feel a boggy or a hardness around the uterus. We may infer this because nearly all of the germs of putrefaction cannot be absorbed into the blood. They are anaerobic and cannot be absorbed or live in fluid, so they are confined to the uterus; but if we find the streptococcus mixed with the putrefactive infection, we may suspect that the disease has extended further than the uterus and that the poison is being absorbed into the system. If we are convinced that the infection has gone no further than the granulation layer at the base of the endometrium, what are we to do? A few years ago it was fashionable to thoroughly curette these cases with a sharp curette. This is bad treatment, for the reason that we already have a wall of protection shutting off infection from the system, and by curetting we destroy this wall, open fresh channels, or weaken the resisting capacity of the structures, and infection, especially if we have the streptococcus present, would then extend more rapidly farther into adjacent structures or into the system. In all forms of putrid infection, where the disease has not gone beyond the uterus, while it is well that we should remove all the retained decidual shreds or blood-clots by means, if we please, of the curette, we must not employ a sharp instrument so as to open any channels that would lead to further invasion. We may with the finger scrape away the septic and broken-down dead tissues, or we may with a blunt curette gently scrape off the structures, irrigating the cavity thoroughly with bichloride of mercury solution and tampon with iodoform gauze; this being followed by gratifying results, because we have not destroyed the protecting barrier, and we have removed the substances in which the bacteria of putrefaction develop and multiply, and have probably removed most of the bacteria, and what we have not removed we may have, by the use of the antiseptic solution, destroyed their vitality, and the iodoform gauze may prevent further development of germs left in the cavity, the vitality of which may not have been destroyed.



By these means in many cases we may rapidly cure our patient, but if we break down the barrier, then the disease goes on, and maybe in a few days this local trouble, against which nature might have finally protected the system, is no longer isolated, and the malignant germs or the products of bacterial growth are not confined, but enter the veins or the lymphatics and destroy life.

If the disease has extended to the parenchymatous structures of the uterus, then what are we to do? Shall the curette be used, and will it do any good? We may safely say that in such cases it will do no good, but it is probable that frequent irrigation into the uterus with bichloride of mercury solution followed by sterilized water may in this form of infection, and also in the form that we have just spoken of, be of decided benefit, because we are constantly washing away or weakening the vitality of micro-organisms that would otherwise produce toxines or ptomaines that continue to poison the system; or we are washing away or weakening micro-organisms that may be otherwise carried outside the uterus and cause further involvement. If the infection has not extended beyond the uterus, most of these cases, if the bowels are kept open with salines two or three times a day, and the vagina irrigated with bichloride of mercury solution, will recover without any intrauterine treatment. But what are we to do if the disease has extended to the peritoneum around the uterus, or in the cellular structures of the broad ligaments, or in the Fallopian tubes, causing either salpingitis or pelvic peritonitis, etc.? I believe that many of these cases may be cured by timely surgical treatment not of a dangerous character. We may open Douglas's pouch or the folds of the broad ligaments, separating adhesions, exposing diseased surfaces, incising pus-cavities and irrigating them with sterilized water, and gently tamponing with iodoform gauze so as to give free drainage. By this means we may prevent further extension of the disease, prevent the formation of abscesses, and encourage speedy convalescence. And this treatment, which has until recently not been mentioned, will be the favorite means of treating acute cases of septic infection, and will be the means by which they will be cured; but if otherwise treated, infection may finally extend to other structures and probably cause systemic infection. In a case where infection is not confined to the uterus, and there is a small or large accumulation of pus, whether the pus be in the broad ligaments, in the tubes, in the ovaries, or inside

the peritoneum, the correct treatment is to open Douglas's pouch, open the abscess sacs wherever they may be, drain away the pus, irrigate the cavity thoroughly with sterilized water, tampon with iodoform gauze, and if we cannot in this way succeed in getting into all the infecting foci, then, if necessary, perform a vaginal hysterectomy, removing the uterus, the ovaries, and the tubes. This treatment and drainage, or drainage with removal of the uterus, will usually cure these patients, provided the infection has not become general. But if we have a case where the acute infection has involved the entire peritoneum, causing general suppuration, then vaginal or abdominal hysterectomy and flushing the abdominal cavity, separating the dense adhesions which bind the intestines together, will do no good, and our patient will die just the same as if we did not perform any operation. While gynecologists and obstetricians recommend laparotomy in cases of this sort, and claim that they have cured them, there is no positively proved case on record, and those cases that they claim to have cured were localized peritonitis in certain parts of the abdominal cavity. Or if we find that the septic matter has gone into the lymphatics or veins and infected the entire system, then all forms of surgical treatment, except to open the pelvic abscesses, is contraindicated. If we find that the septic matter has been carried through the veins to different parts of the system, causing infection of the pericardium, pleura, liver, spleen, kidney, or any structures of the body, sometimes abscesses in the lungs, or in the connective tissue of the body, then all surgical treatment, unless it be to open a pelvic abscess, is contraindicated.

It is difficult to know just when surgical treatment is indicated and when it is not, and at present we are not always able to decide, and may never be. This is unfortunate, because we know that in the worst forms of septic infection, if surgical treatment is to be of benefit, it is at the beginning of the disease; it is while the disease is confined to the pelvic structures, before it has to any considerable extent extended through the lymphatics or veins into the system, or before it has caused general suppurative peritonitis.

There are two ways of performing hysterectomy in septic formations confined to the pelvic structures. One method (vaginal hysterectomy) we have already described and recommended; the other is by abdominal section, opening the abdomen and removing the ovaries, tubes, and uterus, above the pubes. This latter method is

more difficult, more dangerous, and certainly cannot be any more thorough, and results can be no better. So we will advise vaginal hysterectomy in some cases of septic infection involving the pelvic structures, but believe that many of these cases can be cured without removing the organs by simply opening well through the vagina all pus-cavities, and draining with iodoform gauze. We can remove large pus-tubes through the vagina by opening either through Douglas's pouch or by dissecting the bladder in front from the uterus. This morning I removed a pus-tube which was firmly adherent in every part by making an opening into Douglas's pouch, separating the adhesions, bringing the tube down into the vagina.

As you become more experienced in this kind of work you may succeed much better, and what was believed a few years ago could not be done, can in the future be done more easily than by the abdominal route.

## THE TREATMENT OF SCABIES.

CLINICAL LECTURE DELIVERED AT THE POLYCLINIC HOSPITAL, PHILADELPHIA.

BY J. ABBOTT CANTRELL, M.D.,

Professor of Diseases of the Skin in the Philadelphia Polyclinic and College for  
Graduates in Medicine; Dermatologist to the Philadelphia Hospital and  
to the Frederick Douglass Memorial Hospital.

---

GENTLEMEN,—In presenting to you the subject of this morning's talk, the treatment of scabies, I appreciate the fact that the diagnosis as well as the treatment of this skin affection is sadly at fault in many instances, and as I progress in my remarks I will allude somewhat to the symptoms presented, as well as make reference to the diagnosis, so that you may not be led astray.

You should all remember that the cause of this eruption is the ravages of the *acarus hominis*, the so-called itch-mite, and that it is the female parasite that provokes the mischief. After passing from the burrow which was made by its mother it soon becomes impregnated. After impregnation she enters the skin by burrowing in a rectangular manner beneath the surface of the skin, and at about an angle of 45 degrees. This burrowing is in a similar manner to a mole entering the ground. As she progresses in this self-made groove she deposits an egg at regular points. These eggs generally reach the number of six, and seldom go beyond this figure. After depositing the last egg the mother parasite dies and the body remains as a foreign substance. As the time approaches for the first deposited egg to be hatched a pustule is formed at the point of entrance, and through this the young pass to the skin. This parasite meets the male, becomes impregnated, and goes through a similar performance as its mother before it. These manœuvres of the *acarus* cause intolerable itching, for the relief of which the affected person scratches and tears at the skin without gaining any cessation of the sensation, but producing a complication in the form of a dermatitis or eczema which adds to the discomfort. For its point of

attack the acarus selects those portions of the body in which the skin is thinnest and where it may receive the least harm to itself. It may be found upon the sulci of the fingers or upon the palmar surface of the wrist, in the bend of the elbow, and in front of the shoulder or in the crural region. The burrow in which it has its habitat may be seen as a small or large dark streak, which may accept a straight, curved, or tortuous line. The upper edge of this burrow may be noticed to have irregular breaks in the skin, and into this raised portion the effete materials may collect, thus giving it a dirty black color. Usually this line is from one-eighth to one-quarter of an inch in length, but often has been seen to be much longer.

The male acarus, other than impregnating the female, plays a very inconspicuous part in the production of this condition, and therefore needs little, if any, attention at our hands. It may be found occupying some shallow excavation in the skin. The female parasite which causes all the trouble, therefore, will occupy our attention, and, as the description of Radcliffe Crocker is undoubtedly the best that I know of, I will give in detail his description. This author says, "It must be remembered that an acarus is not an insect, but, having eight legs, belongs to the tracheal order of the class Arachnidæ,—viz., the acari." "The female is just visible to the naked eye as a minute, white, shining, roundish body, one-eightieth to one-sixtieth of an inch long (0.3022 to 0.4322 millimetre), and about two-thirds of its long diameter in width. Attached to its conical stumpy legs are four suckers anteriorly, and four setæ or bristles posteriorly, one to each limit; on the back are numerous transverse striæ and scattered lines, with a few short, nail-like setæ; while on the under surface are the legs, a few setæ, and sometimes an ovum." "The larvæ has at first only six legs, and as it is not until after its second or, as some say, its third moult that it is fully developed and has its full complement of eight legs; it, too, burrows a short distance while it is undergoing its moult. When a cuniculus is snipped out with scissors and examined, the ova are found in it in all stages of development, with fæcal and other *débris*, with the most mature ovum at the oldest end of the burrow, and the mother acarus at the other."

In a well-marked case we may look for the condition occupying the hands, between the fingers, around the wrist, at or near the bend of the elbow, around the girth of the body, over the buttocks, upon

the penis in the male and mammæ in the female, and along the inner portion of the thighs, and, in fact, any portion of the body that can be reached by the hands which act as the carrier. After the condition has existed for some time we have in addition to the above-mentioned lesions numerous vesicles and pustules formed which assist greatly in the aggravation of the sufferer. The face is never involved except in children or infants who are at the breast of a mother, whose mammæ show the condition, and from which source the child contracts the affection. It is only in the most aggravated cases that other portions of the body are affected. Usually we are not confronted with the condition in the intrascapular region, or at points above this, nor do we find it occupying the upper portion of the chest or surrounding the neck, but in these most aggravated cases we may find the lesions occupying all portions of the bodily economy. The number of burrows increase in equal ratio with the increase of the number of parasites, and as they may be scattered at very diverse regions there is no telling how great or terrifying the condition may be without you have once been a sufferer. The scratching that is indulged in gives no relief, but intensifies the affection and causes the production of ragades or fissures which show truly the characters of an eczematous eruption. There may be oozing and crusting and the parts may become infiltrated, as observed in a true case of eczema. When these complications arise they undoubtedly throw a mantle over the condition, and errors of diagnosis are likely to occur which cause the use of inappropriate treatments, thus keeping the affected person longer in their agony.

Thus, it can readily be seen that the diagnosis is not always an easy matter, but offers great difficulty in many instances. In any affection of the skin in which the lesions have occupied so great a distribution, it is always wise to suspect that scabies is present. If you will then look carefully for the characteristic symptoms—the presence of the burrow around the fingers, about the wrists, upon the mammæ in the female, and upon the penis in the male—then follow this with an examination into the feelings of the affected persons. Ascertain whether there is itching present and whether there is a history of contagion. If all these points are corroborated, or if only the burrow is found, it will be sufficient to make your diagnosis of scabies.

You must differentiate the eruption from eczema which does not

show the same multiformity of lesions. In eczema the lesions are more likely to congregate in regions, and, in fact, certain eczemas may coalesce and form patches. The itching will not be so diverse in character nor will it be so severe. The patient will not be disturbed to such an extent during the sleeping hours in eczematous conditions.

Pediculosis generally selects different portions of the body for its attack,—especially places where the folds or seams of the clothing touch the skin. The pediculus can always be detected either traversing over the skin or situated in the seams of the clothing in which it makes its habitat.

Contagious impetigo when it attacks a great amount of surface may often resemble scabies, but the characteristic crust with the peculiar discharge which is observed after removal of this crust, and the smooth, glistening surface which is found are enough to differentiate the two conditions.

It is not so often necessary to diagnosticate between this condition and prurigo for the reason that the latter affection is rarely observed in this country. In prurigo the affection is usually observed in early childhood, and generally with an attack of urticaria. Contagion does not occur. Pruriginous lesions are found located on the extensor surfaces of both extremities. Prurigo shows papules or tubercles, while scabies shows vesicles and pustules. The itching character of scabies with the presence of the burrow are sufficient to diagnosticate from syphilis. Syphilis may be shown in the same case, but other symptoms will be present. After being careful in making your diagnosis, and having arrived at the correct solution of the difficulty, you have next to make up your mind what form of treatment is most adaptable to the case presented before you. There are numerous remedies that are said to have a controlling influence upon this cutaneous affection, but it will be your province to select those which have the better effect, as it is not wise to rest content with only one drug that you know will cure, because there are cases in which the best-selected applications fail in their effect. They may fail, not because the drug is not the proper one, but because the strength of the preparation is not sufficient, or they fail on account of the improper manner in which they may be applied by the affected person.

Of the remedies which are applicable in this affection sulphur

has been used for many years either in the form of the sublimed powder in ointment form, or many combinations may be made in which this drug forms the main part. I may mention one of the formulæ which has been very successful in my work in years gone by, and in doing so I will refer to the manner of using it. First of all, it will be wise to remember that we have persons presented to us for treatment in whom the skin is thin and delicate, and in whom you must be careful not to advise a remedy which is exceedingly active for fear of producing some complication which in itself may be adding fuel to the flames. Other persons may apply for treatment in whom the skin is thick and tough and in whom the strongest remedies may be required to effect the desired result. Therefore it will be found advisable, especially until you are acquainted with the state of the skin of the presented individual, to advise the mildest application, and, after becoming thoroughly satisfied with affairs, to then adapt yourselves to circumstances. Wilkinson's ointment, to which I alluded when speaking of sulphur, is made as follows: Sublimed sulphur and oil of cade each two drachms, prepared chalk one and one-half drachms, green soap and lard each one ounce. This is the formula modified by the elder Hebra. It will be found adaptable in those old and chronic cases of scabies which are met with in general practices, but usually it will be found more advantageous to slightly weaken its strength in cases such as will be seen in your private work. I have repeatedly seen cases in which this preparation had been used, and in which some complication, such as eczema, had been induced, thus giving the physician some feeling that an error had been made in his diagnosis, but which had only been the fault of a strong application. Owing to this feeling that complications may arise by the injudicious manner of applying this remedy by the affected person, I have lately discarded its use both in my private and public work. Helmerich's ointment, another preparation containing sulphur, has also a field of usefulness in the treatment of scabies, and I will give you the formula: Sublimed sulphur one drachm, carbonate of potassium one-half drachm, lard sufficient to make one ounce. This also is a strong ointment, and must be applied with caution until you see how it affects the skin. By using this formula in diluted strengths you will be surprised with the many cures that will be effected.

The formula in use mostly in my practice has consisted of some



sulphur preparation, and the one by which I have secured many good results contains the following ingredients: Sublimed sulphur one drachm, and seven drachms of lard or petrolatum; but as this formula alone has often failed to cure the cases which have presented themselves for treatment, I have recently added another ingredient which, in my mind, greatly enhances the value of the preparation. I use the same ingredients as mentioned a few moments ago with a drachm of beta-naphtol to the ounce in the most aggravated cases, but decrease the amount of beta-naphtol in those persons in whom I detect a thin or otherwise irritable skin. In children it will be found advisable to make your preparation correspond to the age of the affected one, and the preparation used in my clinical and private work is usually fifteen grains of each of the ingredients to the ounce of ointment. This may be increased according to the age or the effect of the ointment, but always taking care not to produce any complications. It is almost certain that some form of eczema or dermatitis will be induced by whatever treatment may be adopted, but this can be made very slight by the closest attention to the preparation of your remedial measures. Styrax may be used either in the liquid or ointment form. Of the former we may add about one ounce of styrax to half or one ounce of olive oil, or the preparation may be made more effective by the addition of about one drachm of the balsam of Peru. Ointments may be made with lard or petrolatum in the strength of one or two drachms of the styrax to the ounce of ointment, or we may add some sublimed sulphur to this, as, for instance, one or two drachms of the latter—being governed by the state of inflammation present and the character of skin that you have to treat. Green soap often plays a very important rôle in the treatment of scabies in which there is a tough or thick skin, and the addition of one-half to one drachm to the ounce of ointment often proves curative. Cases may be observed in which it will be found judicious to use a combination of some of the previously mentioned remedies, and you must use your own judgment as to which will be the most beneficial to advise in the case presented.

The application of one of the ointments mentioned is not the only measure that should be adopted, but the manner of previously and subsequently treating your patients must receive the proper amount of attention. Before using the ointment the patient is to be given a bath,—a thorough bath, either in hot water alone or one

to which some antiseptic has been added. It is not advisable to commence the treatment of a case without first removing all *débris* that may have collected upon the skin. The hot bath may be sufficient to do this, but the addition of ordinary soap or a slight quantity of the German green soap will often be found advisable adjuncts. The manner of advising baths in this department is that the patient is to have his bath of thirty gallons of hot water ready at the time he removes his clothing and to then add his green soap to the extent of one ounce to the bath, or, if this is not attainable, owing to his pecuniary circumstances, he is told to get a cheap brown soap. Immediately upon getting into the tub he begins to wet his body and then to lather it well and rub the entire surface in a brisk manner until a red glow is induced, after this he dries himself thoroughly with some soft towel, and then applies the ointment to each and every portion of his anatomy. The application of ointment is to be made every night upon retiring, and in severe cases it may be found advisable to also have a slight or hard rubbing in the morning before going to business. After two or three or, possibly, four days have past, he is to again go through the same process in the bath and not to apply the ointment for twenty-four hours. After the expiration of the limited time he is again to go through the application of ointment process until another three or four days have past, when he again takes another bath, at which time it is best that you make an examination. At the time of this examination, if you are unable to detect any burrows, it may be felt that you have effected a cure; but if you see or feel that burrows are present, you must allow him to again go through the process for a day or two and to again report at your office. If you are now satisfied that you have effected a cure, it will then only be necessary to give him some slight ointment of a mild character to disperse the dermatitis that may have been induced.

It is hardly ever necessary to treat the clothing; but if you desire to be cautious, this may be placed in some superheated atmosphere, which may kill any parasites that may be secluded here. The underwear may be boiled for some time in an ordinary tin boiler, while it will be necessary to cover the outer clothing with some brown paper and then place it in the oven so that all acari may be killed.

## THE PREVENTIVE TREATMENT OF APPENDICITIS.

CLINICAL LECTURE DELIVERED AT THE PITIE HOSPITAL.

BY ALBERT ROBIN, M.D.,

Professor Agrégé in the Medical Hospital of Paris, Member of the Academy of Medicine, etc., France.

---

GENTLEMEN,—The patient we have just seen together, an omnibus conductor, thirty-four years of age, was seized suddenly two weeks ago with a violent pain in the right iliac fossa. On admission to the wards he presented the classical symptoms of appendicitis: distended abdomen, painful tumor in the right iliac region, the pain radiating in different directions; the sigmoid flexure filled with faecal matter; together with high temperature, vomiting, dry tongue, severe thirst, etc.

In view of these symptoms we asked the opinion of a surgeon; especially as our colleague, Tuffier, says that the diagnosis of appendicitis is open to no question, and that the patient ought to be operated on as soon as the crisis is over. A detail that is of importance in this instance is that before this attack of appendicitis our patient had suffered from muco-membranous enteritis. We have therefore to do with a case of appendicitis to which the surgeon lays claim, and I shall take advantage of this occurrence to speak to you about this disorder, to which the medical world is now devoting a great deal of attention, and which is under discussion by many of the learned societies.

If a person unfamiliar with medical matters were to follow these discussions at the present time he would certainly derive a strange impression from them; he would find physicians claiming that surgical treatment is the only one suitable for appendicitis, while, on the other hand, he would note many surgeons refusing to operate on non-suppurative cases of the same disorder.

It appears to me that both physicians and surgeons have wan-

dered from the proper path in this question by leaving out of consideration the causes, or at least one of the principal causes, of appendicitis; since, if I make a possible exception in favor of certain special cases of this disorder, such as those caused by the presence of a foreign body in the appendix, or by the suppuration of a neighboring tissue, I firmly believe that the pathogenesis of appendicitis is nearly always the same, and this idea leads us to the conception of a prophylactic treatment of the disorder.

I consider appendicitis and muco-membranous enteritis, which are generally classed as distinct complaints, as the two ways in which certain cases of dyspepsia react on the intestines. I think I may say that I do not remember a case of appendicitis in which I did not discover the signs of pre-existing dyspepsia, of a special form, characterized by six groups of symptoms.

1. The patients have an excellent appetite, but on examining them you are struck by the contrast between the large amount of food they consume and the wretched appearance they present.

2. The occurrence of crises of gastralgia, very often taken for crises of appendicitis, but from which they differ by their location.

3. Gastric distention, as shown by the characteristic gurgle; this distention is due to contraction of the pylorus.

4. Increased hepatic volume, with exaggeration of the functional activity of that organ.

5. Coprostasis in the different segments of the large intestine; either in the sigmoid flexure, the descending, transverse or ascending colon, or the cæcum.

6. Chemical examination of the gastric juice after a test meal shows exaggerated and abnormal acidity.

In fifty per cent. of such cases the analysis also reveals free albumen that has not been affected by the hydrochloric acid and that still coagulates with heat.

In seventy per cent. of the cases there are present acetic, lactic, and butyric acids, isolated or combined, which take a part in the hyperacidity of the gastric juice.

This form of dyspepsia is what I have called gastric hypersthenia, but other authors mention it under varying appellations.

The idea which I now advance, that appendicitis and muco-membranous enteritis are the two ways in which gastric hypersthenia reacts on the intestines, can be demonstrated by my statistics of the

11011

latter complaint. In 1585 cases of gastric hypersthenia that have come under my observation there was appendicular disorder eighty-two times, and on thirty-three occasions appendicitis was recognized and diagnosticated not only by myself, but by competent surgeons. The relation between the gastric disorder and the inflammation of the appendix consists, in my opinion, in coprostasis; the accumulation of fæcal matter in the cæcum caused by this form of dyspepsia gives rise to the appendicitis, just as its accumulation in the sigmoid flexure and colon produces muco-membranous enteritis. In my fifteen hundred and eighty-five cases coprostasis was noted eleven hundred and six times.

Following out this line of thought I consider appendicitis as the result of seven morbid stages:

1. An arthritic constitution.
2. Gastric hypersthenia, with secondary acid fermentation; as there are a certain number of cases of gastric hypersthenia in which there is no secondary acid fermentation.
3. Coprostasis; but not an ordinary form of fæcal accumulation, as the fæces are not normal as will appear from the following analysis:

Fæces passed in twenty-four hours.

	In the Normal Condition. Grammes.	In Gastric Hypersthenia. Grammes.
Weight of fæces . . . . .	145.	200.
Solid residue at 100° C. . . . .	86.25	60.
Organic matter . . . . .	32.02	52.50
Inorganic matter . . . . .	4.28	7.50
Total nitrogen . . . . .	1.20	3.54
Albuminoid matter . . . . .	7.60	22.12

The weight of albuminoid matter can be obtained by multiplying the total nitrogen by 6.25. Again, the normal reaction of fæcal matter is neutral or alkaline, whereas in gastric hypersthenia it is acid. Furthermore, a person in a normal condition of health throws off in the form of albuminoid matter five per cent. of the nitrogen consumed, whereas in gastric hypersthenia this proportion is 12.61 per cent. Lastly, the mineral contents of the fæces are much larger in gastric hypersthenia, and these mineral elements, phosphates of lime and magnesia, are the residue of alimentary substances.

4. Cæcal reaction; owing to the presence of these products of

abnormal decomposition, the cæcum becomes irritated and reacts by catarrhal inflammation, the appendix also reacting in the same manner.

5. It is evident that the conditions of a catarrhal inflammation in the narrow cavity of the appendix are not similar to those in the cæcum; in the first case the products of the catarrh remain in place, whereas, in the latter they can be carried off.

6. Calcareous infiltration of the cavity of the appendix, calculi in that cavity, then catarrhal reaction of the appendix; in many cases the submucous tissue reacts as well, resulting in occlusion of the vermiform cavity, which, according to Dieulafoy, is a necessary condition.

#### 7. Microbian infection.

In this way a case of surgical appendicitis may be developed, but during the first five stages of our scale the disorder is of a medical nature.

Dieulafoy denies all relation between muco-membranous enteritis and appendicitis, and is confirmed in his opinion by the testimony of Potain and by the statistics of eight hundred cases. Reclus, on the contrary, declares that there exists a frequent relation between the two complaints, and that in one week he had occasion to observe five cases corroborating his opinion. He adds that Potain collected his notes of cases at a period when appendicitis was not yet known. Potain replies that though he did not call it appendicitis at that period, he did know it as typhlitis.

As for me, I think that iliac or colic coprostasis causes muco-membranous enteritis, and that cæcal coprostasis causes appendicitis. In the cases to which I have already referred there was muco-membranous enteritis in nine per cent. of the cases of coprostasis, and in thirteen and a half per cent. of the cases in which gastric hypersthenia was combined with it.

The question has been raised whether the existence of intestinal sand has any connection with muco-membranous enteritis. Out of thirteen cases of intestinal lithiasis which I have followed, two showed muco-membranous enteritis as well. Dieulafoy is inclined to look on intestinal sand as a manifestation of arthritism. A point, however, has to be taken into consideration that has hitherto been neglected: the chemical composition of intestinal sand has not been brought into the debate. Berlioz made a good analysis of it, but did

not put it to proper use, as the analysis did not confirm the idea he was supporting. In my thirteen cases I found phosphoric acid, 13 times; lime, 13 times; magnesia, 13 times; woody and vegetable fragments, 12 times; silica, 8 times; fat, 7 times; cholesterine, 3 times; charcoal, 2 times; sulphates, 2 times; chlorides, 2 times; biliary pigment, 2 times.

The particles of charcoal swallowed with the food, the presence of fragments of wood, the silica which enters the digestive tube with insufficiently washed vegetables, in fact, these entire analyses prove that intestinal sand is not a product of secretion of the intestine, but is of extra-intestinal origin. Muco-membranous enteritis does not, therefore, appear to me to have any connection with intestinal sand, but to depend on a special kind of coprostasis, associated with chemical and mechanical irritation, which enables us to view the relation existing between muco-membranous enteritis and appendicitis in a different light. I found appendicular disorder in 6.71 per cent. of the cases of muco-membranous enteritis; the connection between these two complaints, therefore, and gastric hypersthenia appears to me to be definitely proved.

This discussion is not without its therapeutical conclusions, as it leads us to a prophylactic treatment of appendicitis. Every sufferer from gastric hypersthenia is predisposed to appendicitis; in all such cases, therefore, the cæcum must be watched.

1. Whenever in gastric hypersthenia coprostasis occurs, a laxative should at once be given; not a saline purgative, which would cause catarrhal secretion, but castor oil, calomel, compound senna powder, or the following preparation:

R Socotrine aloes, 2 gms. or grs. xxx;  
Vegetable turpeth,  
Resin of scammony,  
Resin of jalep, aa 1 gm. or grs. xv;  
Extract of belladonna,  
Extract of hyoscyamus, aa 0.15 centig. or grs. ii.  
Fiat mass et div. in pil. No. 50.  
Sig.—One or two at bedtime.

If this means is insufficient, use a rectal injection with an india-rubber catheter; when the liquid has been injected, vary the patient's position so that the different portions of the large intestine can be reached. Gentle massage employed at the same time is advan-

tageous, but it should concern the bowel only, as if it acts on the stomach it will tend to increase the spasm of the pylorus.

2. If there is coprostasis, with a sensitive cæcum and hyperæsthesia of the skin, give castor oil only, and combine it with belladonna to avoid pain.

R Castor oil, 80 gms. or  $\text{℥ss}$ ;  
Extract of belladonna, 0.01 or 0.02 centig. or gr.  $\frac{1}{4}$ .

It is well, moreover, to apply the following ointment to the region:

R Mercurial ointment, 80 gms. or  $\text{℥i}$ ;  
Extract of belladonna, 0.10 centig. or grs.  $1\frac{1}{2}$ ;

and put the patient on milk only.

3. If there is actual pain, give an injection of morphine *loco dolenti*; or else rub the region with a sedative ointment of belladonna, opium, and chloroform. Codeine in full doses can also be used, a pill of a quarter of a grain every eight hours.

If there is severe pain, apply leeches to the spot, purge with castor oil, and then watch the patient to prevent further coprostasis. An enema each morning is also advisable.

Do not resort to intestinal antiseptics. Naphthol, benzonaphthol, salicylate of bismuth, or salol, have caused a great deal of trouble; actual intestinal obstruction has been reported from the crystallization of these substances in the intestine. Intestinal antiseptics are useless, and may be dangerous.

When this has been done, the gastric hypersthenia has to be treated in the way in which I have explained to you in previous lectures.

From what I have said you will see that a suitable regimen, laxative remedies, and rectal irrigation will enable you to prevent appendicitis, and by following this method I cured seventy-nine out of eighty-two cases of appendicitis; in three cases only was an operation found necessary. The first was a waiter in a bar, consequently a man following a most undesirable occupation. Suffering from gastric hypersthenia and muco-membranous enteritis, he was suddenly taken with jaundice, appendicular colic, and generalized peritonitis. Laparotomy was performed, and three large gangrenous and ulcerated abscesses were found, as well as a focus of inflammation at the cæcum. He had allowed eight months to elapse without attention



to himself, and he died from the operation. The history of the other two cases was not unlike the foregoing.

As for the seventy-nine patients who recovered by my treatment, I think I may say that they amply demonstrate its value, even when appendicular symptoms have manifested themselves. You will observe that my method is quite different from that advised and followed at the present time by various authorities on the question. Moizard and Legendre declare that it is criminal to purge in cases of appendicitis. Talamon prescribes purgatives. Lucas-Championnière says that the best way to bring on appendicitis is to give opium.

One word in closing concerning muco-membranous enteritis. Siredey treats it with opium; some one else by frequent saline purges, croton oil, and Fowler's solution; a third physician by nitrate of silver enemata and the intestinal administration of chlorate of potash. As for myself, I first relieve the coprostasis, the cause of the disorder, by castor oil or rectal irrigation, and then I treat the gastric hypersthenia, the cause of the coprostasis. This simple method will give you the best results in muco-membranous enteritis.

Gentlemen, this is what I had to say to you this morning. I think I have shown you that by the side of the surgical there is a medical preventive treatment of appendicitis which, as proved by a long series of cases, will in the majority of instances give you entire satisfaction.

# Medicine.

---

## MYOCARDITIS.

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL, BERLIN.

BY PROFESSOR E. VON LEYDEN,

Professor of Special Pathology and Therapeutics and Director of the First Medical Clinic in the University of Berlin, Germany.

---

GENTLEMEN,—Our clinics during the past month or more have been devoted to pericarditis and endocarditis. The last disease is the so-called “heart-disease” *par excellence*. For it is some lesion of the endocardium that is generally intended when we speak of a heart lesion, and usually some pathological change in this membrane which interferes with proper valvular action. But to-day we shall speak of myocarditis, an affection of the heart muscle itself. It is much more important than either pericarditis or endocarditis, for at most they only interfere mechanically with the heart’s action, while myocarditis attacks the heart muscle itself and palsies the activity of that central muscular power in the vascular system on which all vital processes depend.

The study of valvular disease of the heart, since the discovery of the more exact methods of physical diagnosis at the beginning of the century, has occupied so much attention that myocardial processes have been forced into the background. In the days when the pulse formed the physician’s main standby for the diagnosis of heart affections, it must be confessed that clearer notions of the state of the heart muscle itself seemed to exist than later, when the study of the pulse was more neglected because deemed less important. Of late, again, enthusiasm for the study of the pulse has been aroused because of its value as an index of the cardiac muscular activity, and some of this has come from the work on the subject here in our own clinic.

A good example of how much diagnostic importance may attach to due consideration of the frequency, the rhythm, the tension, and the character of the pulse-wave in feeling the pulse is to be found in the patient before us this morning. While a large, seemingly healthy, strongly built man, he comes to us suffering from dyspnoea on the slightest exertion. His hands and his face show signs of cyanosis; his hands, he tells us, have been swollen, and his feet are at present. He has not had rheumatism, and his present condition is the result of a gradually increasing tendency to shortness of breath that has been coming on for some months.

You would decide at once from these symptoms that it was a heart case you had to do with; yet the eliciting of the ordinary physical signs in the thorax does not prove very satisfactory. His apex-beat is visible (only on careful inspection, it is true), and it is but slightly displaced outward and downward. It may be felt rather easily, though it is distinctly weaker than normal. Percussion shows that the area of dulness is increased, though not very markedly. The sounds of the heart while weak and distant are free from murmurs, except, perhaps, for a suspicious impurity of the first sound.

On the whole, no very definite conclusion as to the condition of the heart could be drawn from what we have found in the thorax, but the pulse tells a very different story. It is weak, small, and rapid. It shows an outspoken tendency to miss beats, and not all of the beats that are felt are of the same strength; it is distinctly arrhythmic, in other words. Here is evidence enough that the heart muscle itself is affected. When we inquire as to the patient's habits of life, we find that he has been a plentiful consumer of beer. He is fat enough to make us think at once of fatty infiltration of the heart; but the symptoms point to more than that. The heart muscle itself has been affected. Probably the etiological factors are two-fold: first, there is the myocardial degeneration from the toxic influence of the alcohol; secondly, there is dilatation of the heart cavities from the large quantities of liquid it has had to move. We have myocarditis and the plethoric heart, which recent writers have called the "beer heart."

With regard to the valvular diseases of the heart, we accept the division laid down for us by the pathological anatomist. In myocarditis there is nothing to enable us to detect clinically the forms that the anatomist describes. Whether it is fatty degeneration or

infiltration that is paralyzing the heart's action we cannot tell, so that the most rational division for us would seem to be one founded on the clinical course of the disease and its etiology.

The most important and most frequent form of myocarditis is that which follows or accompanies the infectious fevers. This myocarditis infectiosa, as it is called, may be either acute or chronic. All the infectious fevers through the toxins which they produce affect the heart muscle more or less. The anatomical change produced by the toxins is the cloudy swelling of the muscle-cells described by Virchow and considered the first stage of parenchymatous degeneration. The sad cases of sudden deaths towards the end of many of the infectious fevers, or even when convalescence seemed well under way, were almost mysterious until this affection of the myocardium was recognized and described.

It was studied first especially in typhoid fever, because the disease runs so long a course that the changes mentioned have a chance to develop so as to be easily visible. Investigations, too, were more frequent and more careful in this disease, because all classes of the community are attacked and patients of the better class sometimes fell victims to this dread heart-failure from degeneration of muscle-substance. Later on it was noted in the other infectious fevers, notably in diphtheria, in scarlet fever, and in pneumonia.

In the severe epidemics of diphtheria that we used to have twenty years ago, sudden deaths from heart-collapse were not at all infrequent. It was not uncommon to have a little patient of six or seven years of age, in whose well being, perhaps, one was especially interested, sicken with a comparatively mild form of diphtheria, from which in three or four days it seemed to be thoroughly convalescent. A good prognosis was given the anxious parents, and they were confidently assured that the danger was over. A little more liberty was perhaps allowed the patient. As the result of exertion there came a disturbance of heart action; the little one weakened and lost its playfulness. In haste the physician was summoned, to find a depressed, often collapsed, patient, with a thin, weak, rapid pulse, and in two or three hours all was over.

I remember in my early practice an artist describing to me the death of his little one under just such circumstances. After days of restless anxiety for the life of his beloved boy, who had been almost constantly with him, there had come the doctor's assurance that all

danger was past. They played together for some time that afternoon as before the boy's sickness, and after awhile there came a sudden weakness over the little fellow. He asked to be put back to bed, but died in his father's arms before he could be placed there.

The story produced such an impression on me that I was always much interested in the heart changes in diphtheria afterwards. Long afterwards I found, and my friend Mosler confirmed my own observation, that there was, in consequence of the cloudy swelling and beginning muscle-degeneration, a dilatation of the left ventricle in these cases that could be detected clinically. As the result of chemical substances in the circulation of the heart itself,—the so-called diphtheritic toxines,—a certain amount of fatty degeneration occurred also.

Besides this parenchymatous change, I have since been able to demonstrate that there is an acute diffuse interstitial myocarditis present often in diphtheria, in which there is a round-celled infiltration of the connective tissue of the heart. This inflammatory exudate lying between the muscle-fibres palsies their contractility, partly by mechanical interference and partly because it leads to parenchymatous change also. This interstitial change, while usually diffuse, may occur at certain points more than others, and so present on section the appearance of multiple foci of inflammatory change. In some specimens these may look not unlike hemorrhagic exudates, slight bleedings into the substance of the heart, but close examination, and especially their reaction to stains, will show that they are masses of round cells.

I have seen such interstitial changes in the hearts of fatal cases of pneumonia and once in a case of fatal rheumatism. The case was that of a girl of sixteen, attacked by rheumatism complicated by rheumatic endocarditis. The inflammation of the endocardium spread to the interstitial connective tissue of the heart muscle itself. The case lasted long enough for the round-celled infiltration to give rise to some new-formed connective tissue, as there were distinct fibrous changes to be noted.

(Plates with pictures of the condition passed around.)

All the infections, as we have said, may give rise to heart trouble, and all of them, we have found of recent years, may involve the myocardium. In all of them, then, even measles, for example, the pulse is to be watched for signs of weakening heart. Here we have

the best index of danger to life. But it is not only when we have the signs of collapsing heart that we are to think of infectious myocarditis. Here more than anywhere else "an ounce of prevention is worth a pound of cure." I have given you in talking of diphtheria the picture of heart-collapse as an artist described it. It comes with just as much suddenness and unexpectedness in other infectious diseases.

Any one who has had much practice has seen it occur in typhoid and in pneumonia; and the sudden death of a seemingly convalescent patient, or the hopeless watch by the bedside of one whom you thought out of danger while he sinks despite every medical effort to arouse his fluttering heart, is one of the disheartening things of medical practice.

Owing to strict rules with regard to our convalescents from fevers, we have not had a death of this kind in the wards for a good while. So long as there remains the slightest tendency towards evening rise of temperature or any acceleration of pulse, patients are not allowed to get out of bed on any pretext, and their sitting up in bed is extremely limited. It is not easy to have patients submit themselves to such rules, for they often believe themselves perfectly well long before the danger is past. They must be persuaded that there is the best of reasons for following strict discipline in the matter.

Some years ago it was not a very infrequent occurrence to be told on coming to the wards for the daily visit that one of our cases of infectious fever was dead. Without consulting any one, they would get up, often only to go to the closet, and would die on the way there or back, or would be found in a state of collapse by the attendant after getting back to bed. Sometimes these symptoms supervened after only sitting up for a while, and fainting often occurred for as slight a reason. I have seen two or three deaths from sitting up too soon after an attack of simple serous pleurisy.

The idea that convalescents often get that they must have exercise is a most mischievous one, that must by all means be gotten rid of. It can only do harm, never good, and is the fruitful cause of many delays in convalescence. Sometimes these sudden deaths in infectious fevers are due to emboli of either the brain or the coronary arteries, for the change in the chemical constitution of the blood induced by the toxins of the disease may cause clotting with con-

sequent production of emboli, but the real cause in most cases is the heart-collapse due to muscular degeneration.

Of such a cardiac collapse there are not many premonitory symptoms. Often there is absolutely no sign until the serious symptoms of heart-weakness itself develop. Sometimes the gallop rhythm of the heart-tones may be noted. But this "bruit de galop" of the French writers is of itself of threatening significance in many cases. Where there is really an organic lesion in the case, some clinicians think that it justifies an unfavorable prognosis; while in another set of cases it lacks serious significance altogether and is the result of a heart neurosis.

From what we have said you can see how serious an affection acute infectious myocarditis is, though at the same time, from our success in preventing serious results by simple prophylactic measures here, you can see, too, that its tendency is to complete recovery. All the infectious fevers affect the myocardium more or less, but the process goes on to complete restoration of tissue and function if excessive claims upon the heart's activity in its weakened state be not made.

Besides the acute, there is a chronic infectious myocarditis. This may be the result of acute myocarditis, especially of the acute interstitial myocarditis of which I have spoken, but it may be chronic from the beginning, due to one of the chronic diseases. Tubercular myocarditis is the best known of these chronic processes, the infection with tubercle bacilli taking place either by continuity through the endocardium or by being carried directly to the place of infection by the blood stream.

Syphilis, too, may undoubtedly produce myocarditis, but one should be extremely circumspect in making the diagnosis of syphilitic myocarditis. Everything that occurs in a syphilitic patient is attributed to syphilis. No matter how obvious other explanations may be, syphilis is apt to be looked upon as the important etiological factor in any new symptoms that develop. I have had a young Swede sent to me by a prominent physician in his own country within the last few days who was suffering from chronic endocarditis. There was a clear history of rheumatism in the case, but his physician could not persuade himself that the syphilis, which the young man had contracted some years ago, did not have something to do with his heart symptoms.

Where alcohol, kidney trouble, and arterio-sclerosis, besides rheumatism, can be excluded in a heart case then one may think of syphilis. Changes in the myocardium due to syphilis may be either gummatous in character or be due to syphilitic endarteritis in the coronary arteries. The changes in arteries described by Heubner as characteristic of syphilis, and which are found so typically in the brain, can be found in the heart. The result in both locations is practically the same,—that is, the blood-supply of the organ is interfered with and degeneration takes place from malnutrition. Acute myomalacia cordis may occur where the affection of the arteries is a rapid one, though usually the changes are more gradual and the slow degeneration of muscle-substance is accompanied by overgrowth of the connective tissue.

Where the affection is a gummatous one, mercurial treatment will cure it if it is taken in time. Where the syphilitic arterial affection is recent, the same holds true. When, however, the properly granulomatous syphilitic process gives place to the fibrous change consequent upon the overgrowth of connective tissue, then all medication is in vain. The patient, permanently crippled in the heart, must be spared as much as possible, must have an unirritating diet and plenty of rest.

Another form of myocarditis is the toxic form that is produced by the action of various poisons. This may sometimes be acute. Nothnagel has described an acute fatty degeneration of the heart in chloroform narcosis. The fat-globules found, however, in the heart-muscle cells in so-called fatty degeneration are not always as significant as is claimed. The condition seems sometimes to be merely a passing one. In carbonic-oxide poisoning there is also a degenerative myocarditis, but this does not develop at once, but comes on some time after the poisoning, just as corresponding changes in the brain and other organs. Prussic-acid poisoning, however, gives acute myocardial degeneration.

Alcohol constitutes the most common poison which produces myocarditis as we know it clinically. Sometimes there are circumstances which modify its action materially. In the case we have had to-day two important etiological factors were at work. The man was an excessive beer drinker, so that, besides the toxic influence of the alcohol causing fatty degeneration of the muscle-fibres, the



necessity of moving the large amount of liquid in the system has stretched the muscle-fibres and dilated the heart-cavities.

Tobacco is another toxic agent that seems to produce myocardial change. In this, however, there are strikingly diverse idiosyncrasies in individuals, though when used in large amounts for long times it is undoubtedly not without its effect on any myocardium. I have never smoked to any extent myself, so that I am not in a position to give you any of the clinical symptoms from personal experience;—perhaps there are those in the room who can. As regards the acute toxic influence of tobacco when the system is unused to it, and which undoubtedly affects the heart, too, if one may judge by the seriousness of personal symptoms experienced, I remember having some experience when much younger, but from your sympathetic faces I should judge that you needed no instruction at second hand as to the clinical symptoms.

It was the fashion in Berlin years ago to attribute a great deal to tobacco in heart affections. Traube consoled himself for years, while laboring under a true organic heart lesion, with the notion that it was a tobacco neurosis, or, at least, that the tendencies to lost compensation were to be thus accounted for. The notion was all the more surprising as he was laboring under an organic kidney affection at the same time, which stood in intimate connection with his heart trouble. Frenzel seemed to have some such opinion about his own case, too. A little tobacco is not bad for most people, but as to what constitutes a little is a matter that divides opinions; I have had a young Russian patient who thought it a trifle to smoke fifty cigarettes a day.

One of the most important changes produced in the heart muscle for the clinician comes from the process known as arterial sclerosis. It usually begins in the aorta ascendens, spreads to the valves, and thence to the coronary arteries. The theory of its exact etiology is not very clear. Just how much an affection of the kidneys is a primary element in it is not yet very well understood. Changes that occur in the myocardium are produced by the involvement in the degenerative inflammatory process of the mouths of the coronary arteries. The lessened blood-supply consequent on this causes fatty degeneration of the heart muscle. When the coronary arteries are suddenly blocked by a bit of necrotic tissue from a degenerated plaque in the aorta, then there is sudden death with the symptoms

of true angina pectoris. As a rule, sudden deaths that occur apart from the infectious fevers are due either to apoplexy or to this blocking of a coronary artery.

These emboli of the coronary arteries are not very frequent, but in sudden deaths in comparatively young people it is well to keep them in mind. I remember a very striking case of the kind here in Berlin some years ago,—striking because of the sad circumstances of the case and in the unique pathological find to account for it. He was a man of thirty-five or forty years who had just been married. He and his bride stopped the night after they were married at one of the prominent hotels here. Shortly after they retired he died suddenly. Owing to the circumstances of the case it came into the hands of the legal authorities, and a careful autopsy was made.

Emboli were found completely blocking both coronary arteries. An ulcerated spot of arterio-sclerosis existed not far away from which a portion or portions of disintegrated tissue seemed to have been torn shortly before. The simplest and most suitable explanation seemed to be that on the projecting bit of tissue a clot was formed which during the excitement of coitus found its way from the aortic wall into one of the coronary arteries, leaving a portion of its length projecting. During the intense heart-disturbance incident to one coronary artery being blocked a portion of this clot was shot into the other coronary.

Sometimes these sudden deaths from emboli of the coronary arteries come at an unfortunate time for the physician in attendance on the patient. If they happen some time after he has given medicine it may seem as though it was a poison case. I remember some years ago one of my students writing for my aid with the legal authorities in just such a case. The patient had had, according to his description, a typical attack of angina pectoris, for which he gave about one-eighth of a grain of morphia salicylate hypodermically. Some time later in the attack his patient died, and the friends insisted that the doctor's injection had been the cause of death. I was glad, as I always am, to be of every service possible to him, and I wrote an opinion for the legal authorities that his treatment had been absolutely correct, that it had been what I might have done myself under the circumstances.

These cases terminate so rapidly and so invariably fatally, how-

VOL. I. Ser. 8.—8

ever, that they have very little interest for the clinician. Where the coronary arteries become gradually blocked there is more to learn, and the study of the clinical picture the cases present is well worth our careful consideration. Long before the modern systematization of the physical diagnosis of thoracic disease, before the exact significance of the symptoms that can be elicited by inspection, auscultation, percussion, and palpation had reduced thoracic disease to a science, the symptom-complex which indicates coronary disease had been recognized.

An English physician, Heberden, pointed out about the middle of the last century that attacks of true angina pectoris were the result of pathological changes in the coronary arteries. His demonstration of the fact was so clear that it attracted a great deal of attention and led to a series of observations which confirmed his conclusions. Before the end of the century the case of John Hunter, the great English scientist, had been diagnosed by himself and by the illustrious Jenner, of vaccination fame, and the diagnosis was confirmed by the post-mortem findings after Hunter's sudden death.

The affection of the coronary arteries in such cases is usually arterio-sclerotic in character. Atheromatous patches occur first in the aorta, and gradually the degenerative process spreads into the coronary arteries, lessening their calibre and partially shutting off the blood-current in the special regions they supply. This gradual diminution of the blood-supply has a very different effect from the sudden occlusion of the arteries. The studies of Erichsen, Cohnheim, Samelsohn, of Schultheis, Kolliker, Porter, and Michaelis, have shown that in animals, after the experimental occlusion of the coronary arteries, the heart-action becomes irregular, ineffective, gradually slower, and finally stops altogether. Some of the same experimenters have shown that partial occlusion of the coronaries leads to insufficient nutrition, probably to parenchymatous and fatty degeneration, to fibrous change, and finally to myomalacia cordis,—that softening of the heart-substance which precedes aneurisma cordis. Of heart aneurism we have heard a good deal of late years, because ambitious diagnosticians have considered the diagnosis of the condition *intra vitam* as a subject worthy their efforts. Too often, however, it runs a course that is specifically symptomless, and is only a phase of a complicated myocarditic picture the details of which it is impossible to single out with certainty.

Apart from any changes in the coronary arteries, however, there are changes in the heart muscle that are of special interest. Frentzel and myself have shown that there is a dilatation of the heart apart from any lesions of the valvular mechanism, and yet not due to an outspoken degeneration of muscle fibres. It would seem as though cardiac fibres could be overstretched and then never regain their normal condition. Such dilatation occurs in myocarditis, too, but it may occur in kidney-disease before there has been any organic affection of the heart itself. As I pointed out some years ago, it may be the result of over-exertion, and such a condition is not so rare in people who do heavy work,—heavy lifting and the like,—and in athletes, on whose circulations calls for intense effort are often made.

For nearly all of these conditions, as I have said, the pulse is the most important symptom. Even where other symptoms would seem to lead to a more exact diagnosis, it is the pulse that indicates the extent of the danger that is at hand. You see how important, then, is the study of the pulse, and how necessary it is that in learning other clinical methods this old-time index of heart activity should not be neglected.

Of the treatment of these conditions in which the heart muscle itself is affected I shall not say much to-day. Digitalis is useful often, but one must be careful in its use, for it is not the healthy heart muscle which is excited to increased activity by it in these cases, but a diseased one, and it may be easily still further injured by undue calls on its crippled powers. People who have such a heart condition must be made to realize that they are cripples in the race of life. They must, where their circumstances permit,—and here is where the real difficulty often comes in,—live quiet, easy, untroubled lives; their diet must be bland and simple; there must be no excitants and no excess in eating or drinking; they must learn, quietly and persistently, what best agrees with them, as well as what disagrees with them. This question of diet in chronic cases is the most important therapeutic means we have at our command. I have found again and again that cases that were going from bad to worse under seemingly judicious treatment improved wonderfully on proper regulation of their diet. But that is a chapter I must leave for another day.

## CLINICAL REMARKS ON A CASE OF ACUTE PERFORATIVE PERITONITIS DUE TO ULCERATION OF THE STOMACH.

CLINICAL LECTURE DELIVERED AT THE ROYAL INFIRMARY, EDINBURGH.

BY BYROM BRAMWELL, M.D., F.R.C.P. (Edin.), F.R.S. (Edin.),

Physician to the Royal Infirmary, Edinburgh; Lecturer on the Principles and Practice of Medicine in the School of Medicine, etc., Edinburgh, Scotland.

---

GENTLEMEN,—The case which I wish to bring before your notice this morning is one of great interest; the patient was admitted to the infirmary suffering from acute peritonitis and double pneumonia, due to the perforation of an ulcer of the stomach; she died eighty-four hours after the occurrence of the perforation. The post-mortem examination showed that there were two ulcers of the stomach (one on the anterior, and the other on the posterior surface of the organ), both of which had perforated, the perforation in one being probably post mortem; the peritonitis was limited to the left half of the abdomen.

The notes are as follows:

Mary McD., aged twenty-two years, a cook, was admitted to Ward 27 on the evening of Saturday, October 7, 1893.

*Previous History.*—The history which was obtained from her friends who brought her to the hospital was very imperfect, and at the time of my visit the patient was so ill that it was impossible to question her at any length regarding the previous course of the illness. We gathered that she had been out of health for more than a year, suffering both from her heart and from her stomach; that some months before the present illness commenced she had vomited blood; that on the evening of Monday, October 2, she went out to see the illuminations in honor of the Duke and Duchess of York; that the following day she was suddenly seized with acute pain in the left

hypochondriac region; and that this was the beginning of the present attack.

After the patient's death we found that this statement was inaccurate, for the attack had not commenced until the morning of Friday, October 6. On the evening of Thursday, October 5, the patient had walked to her father's house. She was not looking well, but made no special complaint. The following day the illness from which she died commenced with sudden acute pain in the left side of the abdomen.

*Condition on Admission.*—At the time of her admission she was extremely ill, and evidently suffering from peritonitis; she complained of pain in the abdomen, and lay with her legs drawn up; the temperature was 101.7° F.; the pulse 130.

When I saw her on Sunday morning she was collapsed, markedly anæmic, and apparently dying. The abdomen was greatly distended and tympanitic; the pain had disappeared; indeed, there was not even tenderness when the abdomen was forcibly palpated. She complained of great difficulty in breathing, and said that she was choking. The temperature was 101° F.; the pulse 144; the respirations 38. There had been no vomiting since her admission.

The percussion note was impaired, and numerous fine crepitations and bronchial breathing were audible over the base of both lungs. There was some cough and the patient was expectorating a scanty quantity of slightly rusty-colored sputa.

A loud systolic murmur was present in the pulmonary area; the heart and lungs were pushed upward by the great abdominal distention.

The difficulty in breathing seemed to be due partly to the distended condition of the abdomen, partly to the commencing pneumonia.

*Diagnosis and Treatment.*—Under these circumstances it was difficult to come to an exact diagnosis. It could hardly be doubted that on her admission to the hospital (on the evening of Saturday, October 7) the patient was suffering from peritonitis. The great tympanitic distention of the abdomen which was present at my visit on Sunday morning was apparently due to this cause. I must ask you to note that the absence of pain and tenderness on pressure did not exclude peritonitis. The pain of peritonitis may entirely

disappear in the later stages of the disease and as the result of collapse; and this patient was, you will remember, collapsed when I saw her.

The sudden onset of the attack, with acute pain in the left hypochondrium, and the subsequent peritonitis at once suggested to me that the case was probably one of perforating ulcer of the stomach. The long duration of the attack (we understood at this time that the illness had commenced on Tuesday, October 3) was opposed to this supposition, for perforative peritonitis is usually fatal within forty-eight hours. Nevertheless, Dr. Douglas (who was then acting as my house physician) and I discussed the advisability of having the abdomen opened, partly with the object of relieving the great distention and the difficulty of breathing which was threatening to prove fatal, and partly with the object of determining whether the inflammation of the peritoneum was the result, as we supposed, of a perforation of the stomach; and, if so, of having the ulcer sewed up. We decided against the operation for the following reasons:

1. The condition of the patient was so grave that even granting that a perforating ulcer of the stomach was the cause of the peritonitis, an operation would have been useless.

2. The double pneumonia was a very serious complication.

3. The diagnosis was by no means clear. The fine systolic murmur, the double pneumonia, and the peritonitis were suggestive of ulcerative endocarditis, or, at all events, of a septic condition unfavorable to any operative procedure.

4. And finally, the great distention of the abdomen would probably have given rise to some difficulty. I have seen more than one case of intestinal obstruction in which, after opening the abdomen, the surgeon has had very great difficulty, owing to the great flatulent distention, in getting the intestine back into the abdomen.

Having decided that it was useless to call in a surgeon, we determined to relieve the abdominal tension by puncturing the intestines with a fine trocar, and allowing some of the flatus to escape.

But on returning to the patient with the object of carrying this procedure into effect, we found that the breathing was so much relieved that we thought it best to leave well enough alone and to wait, at all events, for a few hours. I had directed, after examining the patient, that she should be propped up with pillows and that a dose of brandy should be administered. The effect of this procedure was

to allow of the escape of a considerable amount of flatus by the mouth, and to give great relief to the breathing.

A mixture containing menthol, aromatic spirits of ammonia, and spirits of chloroform was therefore prescribed.<sup>1</sup>

The subsequent progress of the case is shown by the following notes:

*On the evening of Sunday, October 8*, the patient's general condition was but little changed. The distention of the abdomen was less marked. The temperature had fallen to 100° F., and the pulse to 124; the respirations had increased, and numbered 42 per minute.

*On the morning of the 9th*, although the patient stated that she felt very much better, I could not see any improvement. The temperature was 99.4° F.; the pulse 130; the respirations 48; the cough was frequent and distressing, and there was a good deal of rusty-colored expectoration.

The pneumonic consolidation had not extended, but the coarse crepitations and bronchial breathing were more marked over the left base than on the previous day. The abdominal distention was considerably less, the result of diarrhoea; through the night the patient had had seven loose motions, moderately copious in amount and accompanied with the passage of much flatus.

*On the evening of October 9* the patient died.

The following day we obtained more definite information as to the previous history. We learned, as I have stated above, that the patient had walked to her father's house on the night of Thursday, October 5, and that she had been suddenly seized with acute pain in the abdomen on the morning of Friday, the 6th. We also ascertained that some months previously she had been treated in the Harrogate Hospital for ulceration of the stomach and some affection of the heart (in all probability anæmia, for her father told us that she had suffered from bloodlessness); that in February she had returned to Edinburgh, and, after staying with her sister for a few weeks, that she had taken a situation and had fulfilled her duties as a servant up to the time of the attack.

The *post-mortem examination* was made on Wednesday, October 11, 1893, by Dr. Leith.

---

<sup>1</sup> I have found this a most useful mixture (menthol, grs. iv to viii; spiritus ammoniæ aromaticus, spiritus chloroformi, aa ʒi. M. Sig.—A teaspoonful when required) for the relief of flatulent distention of the stomach.



The abdomen was considerably distended with flatus. On opening the cavity there did not appear to be at the first glance any peritonitis, but more careful examination showed, when the omentum was turned aside, that the whole of the left side of the peritoneum was covered with a thin layer of recent lymph. The sharp limitation of the peritonitis to the left half of the abdominal cavity was a remarkable feature of the case.

The stomach was considerably dilated and distended with flatus. Its upper surface was glued to the under surface of the liver and the front wall of the abdomen by a thin layer of recent lymph.

On separating the stomach from the adjacent parts, to which it was loosely but closely attached by the thin layer of recent lymph, a clean-cut perforation was seen in the anterior wall; the aperture in the peritoneal coat was circular in shape, in size rather less than a three-penny piece, and situated half an inch from the greater, three and a half inches from the lesser, curvature, and five and a half inches upward and backward from the pylorus.

A second perforation, rather larger than that on the anterior surface, and the margins of which were less cleanly cut, was found, when the stomach was removed, on the posterior surface of the organ; it was situated almost immediately below the ulcer in the anterior wall, one and a quarter inches from the lesser curvature, and about five and a half inches from the pylorus. The margins of the perforation on the posterior surface of the organ were somewhat thickened, ragged, and shreddy-looking. Dr. Leith agreed with me in thinking that the ragged and shreddy condition of the edges was suggestive that the perforation in the case of this ulcer was a post-mortem condition due to the action of the gastric juice.

On opening the stomach the perforations in the peritoneal surface were found to be directly continuous, with two typical simple ulcers; the mucous membrane which formed the margins of the ulcer in the posterior wall was injected.

The heart and pericardium were healthy. There was no pleurisy. A localized patch of pneumonia was present in the lower lobe of each lung; that in the left lung was more extensive than the right.

The heart-muscle was soft and pale, and the ventricular cavities somewhat dilated; but there was no valvular lesion. The anæmia from which the patient had previously suffered was in all probability the cause of the (slight) changes in the heart.

The other organs were free from disease.

*Remarks.*—Such are the facts of the case. It presented several points of clinical and pathological interest. In some of its features it was typical, in others very exceptional.

The patient was a servant girl, aged twenty-two; she had for more than a year suffered from bloodlessness, and had been under treatment in the Harrogate Hospital for ulcer of the stomach and heart symptoms. As you all know, simple perforating ulcer of the stomach is especially apt to occur in young female servants who are chlorotic. In these respects the case was typical.

It was quite exceptional in its long duration after the occurrence of the perforation, and in the remarkable way in which the peritonitis, which had resulted from the perforation, was limited to the *whole* of the left side of the abdominal cavity.

The case shows the great difficulty of coming to an exact diagnosis in the absence of a clear and reliable history. Had we known that the disease had only commenced on the morning of Friday, October 6 (instead of, as we were led to suppose, on the morning of Tuesday, the 3d of October), the probability of the case being one of perforating ulcer of the stomach would have been greater. But even if we had known the exact day on which the attack had commenced, I doubt whether we could have advanced much further than we did in the direction of a positive diagnosis. The double pneumonia was a complicating element.

On reconsidering all the facts of the case, in the light of the more definite history which was obtained after death and of the conditions which were found post mortem, it is, I think, clear that we acted rightly in deciding against any operative interference. As I have already stated, the extreme condition of collapse, and the double pneumonia which was present, together with the other clinical facts which I have previously mentioned, precluded operative interference, and even if the abdomen had been opened, little or no advantage would have been gained; for the peritonitis was very much localized and entirely confined to the left half of the abdomen. The perforation in the anterior wall of the stomach, which was obviously the cause of the peritonitis, was sealed up by the recent adhesions. Had the patient lived, a natural, though perhaps only a temporary, cure would probably have taken place. If the abdomen had been opened, it might have been difficult to replace the intestines.

I say nothing regarding the presence of the second ulcer which was found on the posterior surface of the stomach after death, for, as I have already remarked, I am disposed to think the perforation of this ulcer had taken place post mortem.

Perforation is one of the great risks to which every patient who is suffering from simple ulcer of the stomach is liable. The rupture of an ulcer into the general cavity of the peritoneum is usually followed by intense general peritonitis, which generally proves fatal within forty-eight hours. Recovery does no doubt occasionally occur after such an accident, but it is so extremely rare that for practical purposes it may be left out of account. An *early* operative procedure is the only means which is likely to prove successful in cases of this kind. Opiates and other palliatives are useless. Unfortunately, many of the cases occur in private practice, and under circumstances which to all intents and purposes usually preclude successful surgical interference. The operation is not an easy one, and would, I suppose, be very difficult or impossible without skilled assistance.

But even in those cases which occur in a hospital, and in which the operation is at once performed,—and satisfactorily performed,—the pathological condition of the stomach may prevent a successful result. In this particular case, for example, even if the ulcer on the anterior wall could have been successfully dealt with, the ulcer on the posterior surface (the presence of which was entirely unsuspected) would have remained; it would in all probability have given rise to further difficulty, and might very likely have interfered with the success of the operative interference.

The peritonitis which results from the escape of the contents of the stomach into the abdominal cavity is usually very virulent and septic. The remarkable way in which the peritonitis was in this case limited to the left half of the abdomen, and the comparatively slight intensity and aseptic character of the peritonitis, were probably due to the fact that when the perforation of the ulcer in the anterior wall took place the stomach was distended, or, at all events, that little or nothing except flatus had escaped from the stomach into the peritoneum.

I may further say that my experience leads me to think that not only is *early* operative interference of great importance, but that the success of the operative procedure depends to a considerable extent upon *the rapidity with which the operation is performed.*

## **WEAK HEART; GASTRECTASIS FROM PYLORIC SPASM.**

**CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.**

**BY CHARLES G. STOCKTON, M.D.,**

**Professor of the Principles and Practice of Medicine and Clinical Medicine in the  
Medical Department of the University of Buffalo; Attending Physician  
to the Buffalo General Hospital.**

---

GENTLEMEN,—This case illustrates a condition rarely spoken of in text-books, and apparently but little studied. The affection is most often seen in athletic men engaged in some occupation requiring severe repeated efforts at lifting or straining, and in my experience is almost limited to lumbermen who are in the habit of moving logs in the woods or in mills.

This patient is forty years old, an American, married, the father of healthy children; he is by occupation a lumberman in Pennsylvania; he is a man of good habits, and has escaped previous illness, save an attack of influenza in 1889. His present illness dates back several months, and made itself gradually apparent. He suffered from a sense of lassitude, became easily fatigued, but persisted in his work. He soon discovered that before his day's work was done there appeared regularly a sense of great depression and pain located in the lumbar region. Later on he became somewhat giddy and short of breath after severe exertion, and later developed dyspeptic symptoms attended with constipation. The urine has been decreased in amount for some time. You discover that the man shows loss of weight; his face looks haggard and shows depression; his complexion is muddy, and the skin shows a blending of pallor and the lividity of cyanosis. The pulse is rather infrequent, beating sixty to the minute, and is extremely weak and compressible. It is the pulse of very low tension, and speaks of poorly filled vessels and feeble cardiac contraction. After putting him through a little active exercise I perceive that the pulse becomes very frequent and the patient complains of

thoracic discomfort. You will observe that the hands are livid as they hang at his side, but as I raise this one the color rapidly fades away and the pulse has quite disappeared from the uplifted arm; the hands are cold and clammy, and he complains of cold feet.

On physical examination the cardiac impulse cannot be seen, and can scarcely be felt on palpation; percussion reveals a slightly increased area of cardiac dullness. On auscultation the first sound at the apex is scarcely audible, and lacks the usual muscular quality. The second sound is comparatively intense and high pitched. At the base the aortic sounds are very feeble, but the pulmonary second sound has considerable intensity and is high pitched; there is a complete absence of bruit. The respiratory sounds are somewhat diminished over the bases of the lungs, but in other respects the chest seems normal.

The man complains of a poor appetite and of dyspeptic symptoms, and the tongue is coated. On making percussion it is found that hepatic dullness exists an inch below the free border of the ribs. Nothing else is found wrong by physical examination. The man voids from twelve to sixteen ounces of urine in twenty-four hours, dark in color, of a specific gravity of 1028, acid, containing an abundance of urates, fourteen grammes of urea, an appreciable reaction to indican, and an absence of albumen and sugar. Nothing of importance is revealed by the microscope.

This makes up a picture of cardiac asthenia, and the failing circulation, in my judgment, accounts for all the symptoms that the man presents.

The backache is not an invariable, but an extremely common, complaint of these patients, the explanation of which is not altogether clear; possibly it depends upon the over-distention of the kidneys with the venous blood, a view which finds some support in the scanty and heavily loaded urine. I have already called attention to the symptoms of backache and weak heart in an article published in the *Buffalo Medical Journal* in August, 1895.

My attention was particularly called to these cases after the epidemic of influenza that prevailed in this country in 1889, and most, although not all, of my patients had been victims of that disease. I do not feel sure that influenza is an etiologic factor, but I am inclined to believe that it is in some cases. This condition appears to me somewhat different from that described by Dr. Da Costa as oc-

curing frequently in soldiers who suffered heart-strain by forced marching during the war. In the instances of ordinary heart-strain that I have studied there was apparently greater nervous exhaustion than is present in the class to which this patient belongs. Fortunately treatment accomplishes much for these cases, but unfortunately the course that is necessary for recovery is very trying for laboring men accustomed to an active life in the open air.

I shall direct this man to be kept constantly in the horizontal position in bed until his heart is strong again. This will require weeks, perhaps months, of self-denial on the part of the patient, but we may find some interest in other items of treatment to be mentioned.

He should have daily massage treatment, and one-half hour of general faradization. He may be raised long enough to have a cold spinal douche every morning. His diet should be moderate in amount, nutritious in character, and an abundance of water should be taken daily. For medication I place the chief reliance in *nuxvomica*, given in large and increasing doses of the tincture as recommended by Musser, until the physiological effects are experienced. Not infrequently a patient will take from fifty to one hundred drops of the tincture three times a day before this result is obtained. It is rarely necessary to give other forms of cardiac stimulants, although in some instances I have found it advisable to add *convallaria* or *digitalis* for a short time. Attention should be given to procuring daily and free evacuation of the bowels. Under this plan of treatment cases have invariably progressed favorably.

As might be expected, after two or three months patients resent the confinement and are likely to return to their active habits too soon. They are very apt to retain most of the benefits that they have gained unless the work is too arduous.

During the past two winters I have applied the Schott baths for a short time in several such cases with marked benefit. I have found it advisable to omit most of the resistive movement often so essential in this useful form of treatment.

I have gone into this matter with some particularity because I find these cases constantly overlooked, and because of the belief that the continuation of the cardiac asthenia leads to an irreparable weakness and a useless life.

## GASTRECTASIS FROM PYLORIC SPASM.

This case illustrates a definite, but not well understood functional disease of the stomach that often produces symptoms suggesting a structural disease, symptoms that are sometimes very serious in character.

This man is thirty years old, single, and has been employed as a maker of bicycles. His family and personal histories are uneventful, with the exception of dyspeptic symptoms, from which he has suffered for years. During the past twelve months these have become exaggerated, and during the past three months so severe as to disqualify him for labor. The man weighs one hundred and thirty pounds, and has lost twenty pounds during the past six months; he appears feeble, anæmic, and depressed. You observe the appearance of despondency and apprehension in his countenance. His skin is sodden and rough and his muscles flabby, speaking of poor nutrition. The tongue is heavily coated and the breath is foul; the bowels are constipated, the dejecta dark colored and scanty; and the urine is diminished in quantity, dark in color, and shows a sediment that proves to be urates. It is acid in reaction, specific gravity 1026, and contains uric acid and calcium oxalate crystals. The patient complains of gastric distress, a sense of heaviness and distention, and sometimes actual pain. He has eructations of gas, sour stomach, and occasionally regurgitates fluid sometimes very acid, at other times bitter in taste. Occasionally he has nausea, and of late he has vomited, at intervals of twenty-four or forty-eight hours, large quantities of stomach contents, for the most part in solution. Following these attacks of vomiting he has greater comfort, and his appetite, which is capricious, is at these times much improved.

The physical examination develops no evidence of thoracic disease. The abdomen now appears natural, but on the day of admission, two weeks ago, there was some prominence of the abdomen, and this was proved to depend upon dilatation of the stomach. Palpation developed splashing sounds from the ensiform to a point midway between the umbilicus and the pubes, reaching from the left flank to a point some distance to the right of the median line. Over the corresponding region a somewhat tympanitic note was elicited on percussion, and auscultation with the stethoscope showed an active gastric peristalsis. The stomach-tube was passed twenty-eight inches beyond the teeth, and there was removed by the expression

method one and one-half litres of semi-fluid stomach contents, acid in reaction (80), and an abundance of hydrochloric acid (20). The combined chlorides were present in large amounts. There was a marked biuret reaction, showing the presence of peptones; there was no lactic acid, but the odor of the other organic acids was noticeable.

Following lavage the man's symptoms were greatly relieved, although he still complained of uneasiness, and at times of pain. It was found that the stomach did not empty itself, and lavage was necessary once in twenty-four hours for the removal of the accumulated stomach contents. In other words, there was present food stagnation, that cardinal symptom of dilatation of the stomach.

The question now arose as to the nature of the obstruction that prevented a stomach having active peristaltic movements from emptying its contents onward. The most careful palpation failed to discover any tumor or induration at the pylorus. The natural history of the case, the man's age, and the character of the stomach contents each argued against malignant disease. A chronic peptic ulcer with resulting cicatricial stenosis, located at the pylorus, might have accounted for the condition. In ulcer marked localized tenderness and pain would be expected. In this case there is slight tenderness in the pyloric region, but the pain has not been localized, nor is it made worse by eating. At no time has there been hæmatemesis. Under the circumstances gastric ulcer could not at that time be excluded. The condition that seemed most probable was that of pyloric spasm, the result probably of the irritating quality of the stomach contents. There must be some other factor leading towards the presence of spasm of this part besides the irritation before mentioned; just what this is cannot be determined; it probably depends upon some peculiarity of the nervous system.

The reasons for the hyperacidity of the stomach contents have also to be found. Without going into the etiology of gastric hyperacidity, it is enough to know for the present that it exists.

Concluding that we had to deal with spasm of the pylorus, the man was ordered a milk diet and the administration of the following prescription every two hours:

R Bismuth subcarbonate,  
Sodium bicarbonate,  
Light magnesium carbonate, aa 1 gramme. M.



This mixture was administered stirred in a little water. Each morning the stomach was emptied and thoroughly irrigated with a saturated solution of boric acid. At first the stomach was found to contain a considerable amount of the milk taken during the preceding day.

Two weeks have now elapsed since beginning the treatment, and the man is at present allowed raw eggs, and once each day some scraped beef and stale bread. Notwithstanding this increase in diet, less and less food is found present at the morning lavage, and the symptoms have progressively diminished. The stomach is still dilated, but I am satisfied that the greater part of its contents now pass the pyloric ring, still meeting with moderate, and occasionally for a few hours, complete obstruction. These factors are very significant, and lead me to believe that there is little or no stenosis at the pylorus save that occasioned by spasm, and that under the soothing treatment and the unstimulating diet this contraction has so far diminished as to allow a more natural progression of the alimentary stream.

It seems improbable that this rapid improvement would ensue if there was present an active ulcer causing the spasm, and it certainly would not follow if there was present cicatricial obstruction from a past ulcer; in other words, partly from the examination of the patient, and partly from the results of the two weeks' treatment, one is able to say that this is an instance of dilatation of the stomach secondary to spasm of the pylorus, and that the present relief has been obtained by the diet and the treatment adopted.

Judging from experience in previous cases, it may be prophesied that the patient will make a complete recovery. He must hereafter practise no little care in the selection of his diet, and he must continue the use of the lavage with sufficient frequency to prevent the over-accumulation of food and to ascertain the state of his digestion. It is a good plan to advise the patient to continue the use of antacid and sedative remedies for a long period. As soon as practicable the diet should be somewhat stronger than that now admissible. Altogether the patient shows improvement in his general health, and if all goes well he can look forward to returning to his employment at the end of four weeks.

## CERTAIN POINTS IN THE DIAGNOSIS OF VARICELLA.

CLINICAL LECTURE DELIVERED AT THE CITY OF GLASGOW SMALL-POX HOSPITAL.

BY ROBERT STEVENSON THOMSON, M.D., B.Sc.,

Visiting Physician to the City of Glasgow Small-Pox Hospital, Scotland.

---

GENTLEMEN,—Varicella is so generally regarded by authors and practitioners as a trivial ailment, that I feel some apology is necessary for bringing it before you as the subject of a clinical lecture. There is, of course, no question as to the trifling character of chicken-pox when considered apart from other diseases, but at a time like the present, when the city of Glasgow is threatened with an epidemic of small-pox, varicella assumes a greater importance, the result of the close resemblance between the characters of the eruptions of the two diseases. In the course of my experience in this hospital<sup>1</sup> I have become acquainted with more than one instance in which the failure on the part of the practitioner to distinguish between varicella and variola has resulted in consequences of the most serious kind. In one case a young girl who manifested a vesicular eruption of a sparse character was diagnosed by the medical man in charge as a case of varicella, and the mistake was only discovered when eight young adults were attacked by symptoms of a more or less characteristic kind, which were followed in the course of a few days by the eruption of variola. Fortunately, none of the patients died, but subsequently one was badly scarred by the eruption. This incident will impress upon you the serious results which may follow on an error of diagnosis of the kind referred to.

A reference to any of the text-books on medicine will acquaint you fully with the points, a consideration of which is supposed to make the diagnosis of varicella comparatively simple; as a matter of fact, however, under certain conditions it is one of extreme diffi-

---

<sup>1</sup> Both varicella and variola are admitted to the City of Glasgow Small-Pox Hospital. All patients suffering from the former disease are vaccinated on admission.

culty, and I venture to think, apart from prolonged observation, a practical impossibility. Some years ago a child of three was admitted to the hospital certified to be suffering from chicken-pox. This child had been successfully vaccinated at the usual age; had no history of prodromata, and presented a very sparse eruption of vesicles on the trunk and extremities. After a careful examination of the history as well as of the eruption, and a consultation with my former assistant, Dr. Marsh, whose experience of variola and varicella was considerable, the opinion of the gentleman who certified was accepted. Daily observation gave rise to no suspicion that a mistake in diagnosis had been made, but on the fifteenth day from the date of the child's admission to the hospital her father and brother were received both suffering from undoubted small-pox. No apology is necessary for a mistake such as this, when we remember the close resemblance which the eruption of varicella sometimes bears to that of variola, and how few opportunities practitioners have of studying the characters of the latter disease.

You will judge from what has been said that my remarks on the diagnosis of varicella will be more or less destructive, and to illustrate these I have selected this patient who has just been admitted to the ward.

Varicella, we are told, is a disease of childhood, and some writers tell us that it never occurs in advanced life. In point of fact, varicella does occur among adults, although rarely, and among young adults, say from fifteen to twenty years of age, it is not uncommon. The age of our patient is seventeen, and he furnishes an example of what would be called an almost typical case of chicken-pox. In this instance there has been no difficulty about the diagnosis, and I may say here that in general but little exists in distinguishing the more marked cases of variola and varicella. Those on the borderland alone present any difficulty. The absence of prodromal symptoms in varicella is generally stated to be an important diagnostic point. In the present instance, unfortunately, we have no record of the patient's temperature prior to admission, but he tells us that the attack was ushered in by feelings of general malaise, severe headache, and feverishness. His temperature, on admission, however, was normal. This history may be supposed to be an exceptional one, but in reality it is the history most commonly given by young adults who enter the hospital suffering from this disease. Among

younger children such a history is generally wanting, in some cases, no doubt, because prodromal symptoms are really non-existent, or slight, but in many instances the supposed absence is the result of faulty observation on the part of the child's guardians. Our experience in the hospital has been that when children contract chicken-pox while under careful observation, the temperature being taken as a matter of routine, a prodromal stage is invariably present. The length of this in varicella is generally supposed to be of some diagnostic value, as it is in most cases considerably shorter than that of variola. On inquiry, however, we find that in the case of our patient the eruption appeared on the third day of the illness, so that here we lose any support which might have been derived from the shorter duration of the pre-eruptive stage. While the eruption of varicella has usually a somewhat irregular distribution, it is supposed to possess no marked selective affinity for the face, and comparatively little for the limbs. This is true in a limited sense only. In the case of our patient the eruption is present on the face, though it is certainly not so thickly set as on the trunk. In many cases, however, and especially in those on whom the eruption is very sparse, there is but little difference in its distribution on the face as compared with the rest of the body. It should also be remembered, however, that in some cases of modified small-pox, in which the eruption is thinly scattered over the surface, the face may not be specially involved, and, in fact, may present fewer eruptive elements than an equal area of skin elsewhere. On further examination of the eruption on our patient, we notice a few elements on the palate and on the palms, while on the sole of the right foot there is a single large and well-developed vesicle. Here, again, the so-called characteristic distribution of the eruption fails to assist us in our diagnosis, for it is usually stated that in this disease an eruption on the palms of the hands, soles of the feet, and palate is rare. So far as my observation goes, an eruption of a very sparse and limited character is by no means uncommon in these situations, more especially among older patients.

The cases quoted at the beginning of my lecture have already prepared you for the statement that the appearance of the individual eruptive elements is not invariably characteristic. As I have already remarked, it is quite unlikely that even a superficial observer could mistake a typical case of varicella in which the vesicles sur-

rounded in their early stage by a narrow rose-colored areola are themselves small, transparent, spherical, and non-umbilicated, lacking in body, superficial, easily ruptured by slight pressure and readily collapsible on puncture. When all these characters are present the diagnosis is easy; but when, as in the case of our patient, we find that the more advanced elements of the eruption are of considerable size, present milky contents, are depressed in the centre, have considerable body (especially on the palms, soles, and limbs), are moderately resistant to pressure, and do not collapse easily on puncture, we at once feel that our basis of diagnosis, if we depend upon the physical characters of the eruption alone, is insecure. But even a superficial examination of the eruption in the present instance will reveal certain general characters upon which we are able to place considerable reliance as aids to diagnosis. You will notice that we have to deal with what I may call a multiform eruption,—an eruption which presents similar characters on every part of the body. An examination of the face, scalp, trunk, and extremities reveals the existence side by side of small red papules, and well-developed vesicles with milky contents, which vary greatly in size and are more or less irregular in outline, while here and there the eruption has undergone complete desiccation with the formation of thin, reddish-brown, superficial crusts. The most important condition to which I would call your attention is the presence of these crusts after the lapse of so short a time from the first appearance of the eruption. The patient first showed signs of illness on the 10th instant. During the night, between the 12th and the 13th, the rash made its first appearance, and now, about thirty-six hours later, we notice that a considerable proportion of it is completely crusted. To my mind this early crusting is the most important diagnostic distinction between varicella and variola, and is a condition which I have never observed in the eruption of the latter disease. The longer duration of the papular and vesicular stages of small-pox will account for this. In varicella both these stages are passed through with great rapidity.

Of less importance, in my opinion, is the contemporaneous presence of papules and vesicles on distant parts of the body. For though in variola there is a general progression of the eruption from the upper part of the body downward, yet not infrequently we find, as a result of local irritation, or other less obvious cause, that the

rash may show itself earlier on certain portions of the lower extremities than upon those parts where, from general experience, we would naturally expect to find it first. This anomalous distribution of the eruption is more frequently seen in modified than in unmodified small-pox, and in the former I have frequently noticed on the lower extremities well-advanced vesicles set side by side with papules, while on the face and upper portion of the body the individual elements presented practically the same stage of development.

Occasionally vaccination enables us to form a differential diagnosis between variola and varicella. This aid, however, cannot always be relied on. It is perfectly true that a person suffering from modified small-pox is insusceptible to the influence of vaccination, but it must be remembered that a very considerable proportion of the patients suffering from chicken-pox are likewise insusceptible, chiefly as a result of that operation having been performed within a comparatively recent period.<sup>1</sup>

In addition to these characteristics, which must constitute the principal factors in diagnosis, may be mentioned "pre-variolar" rashes, sore throat, and umbilication of the vesicles, all of which are regarded as more or less diagnostic of small-pox. In varicella we not infrequently meet with a pre-varicellar eruption, always, so far as my experience goes, of a diffuse erythematous character. In small-pox such an eruption is frequently a very grave prognostic sign, and hence a want of knowledge of the former circumstance may lead to awkward mistakes. The fact in itself gives no positive assistance one way or the other. The absence of sore throat in varicella is of some little value in framing a diagnosis, in view of the fact that in small-pox sore throat is nearly always present, even in the mildest cases. Yet, as we see in the case of our patient, faucial catarrh and an eruption on the palate may occur occasionally in the former. Umbilication of the vesicles is frequently a source of difficulty, more especially to those members of the profession who have had but little opportunity of observing the eruption of variola. More than once I have been told that a certain case could not be one of small-pox, because the elements of the eruption showed little or no sign of umbilication. In point of fact, in a very considerable number of cases of well-marked small-pox umbilication is never

---

<sup>1</sup> In the case of the patient who formed the subject of this lecture, revaccination was completely successful.

present at any stage in the evolution of the eruption. On the other hand, while true umbilication is exceptional in chicken-pox, a pseudo-umbilication, such as can be seen in our patient here, is almost always present when the pocks attain even moderate dimensions. This appearance is probably the result of absorption, which causes a collapse of the central or oldest part of the vesicle.

To conclude: The points to be borne in mind in forming a diagnosis of varicella, given in the order of their importance, are: the early vesiculation and crusting of the elements; the contemporaneous presence on various parts of the body of papules, vesicles, and crusts; the irregularity in size and shape of the pocks, most noticeable on the trunk; the irregular distribution of the eruption, which is found most abundantly on the trunk and scalp, and more sparsely, as a general rule, on the face and limbs; the age of the patient; the absence of eruption, especially in young children, from the palms, soles, and palate; the absence or short duration and trifling nature of the pre-eruptive stage; the absence of umbilication of vesicles in the earlier stage of the rash; the condition of the patient with regard to vaccination; and, lastly, the absence of a pre-eruptive rash.

The diagnosis of varicella, therefore, depends not upon the presence of any single diagnostic character, but upon a consideration in doubtful cases of all the points I have referred to, not only of the eruption itself, but likewise of the general symptoms manifested by the patient.

## ENLARGEMENTS OF THE SPLEEN; FIBROID PHTHISIS; ENDOCARDITIS.

CLINICAL LECTURE DELIVERED IN RUSH MEDICAL COLLEGE.

BY NORMAN BRIDGE, M.D.,

Professor of Clinical Medicine and Physical Diagnosis in Rush Medical College,  
Chicago, Illinois.

---

### ENLARGEMENTS OF THE SPLEEN.

GENTLEMEN,—This patient has been sick for something over ten years. He is thirty years of age. When he was eighteen or nineteen he was a railroad man, as he is now, and his work lay in the State of Indiana. During 1878-79 he had from time to time attacks of ague. In 1879, he was confined to his bed for several days with remittent fever. After that he had a recurrence of the chills from time to time,—that is, a profound chill, followed by a high fever, which would last a number of hours and disappear. He would have to stop his work and be under the direction of a physician. As soon as he became a little better he would resume his work, only to have the same thing occur again and again. This continued for about two years. He had, at the end of that time, what was called malarial pneumonia. I have no reason to doubt the diagnosis. He then left the State of Indiana, went into Wisconsin, where he remained perhaps a year, and became somewhat better. Most of the time since then he has lived in Illinois.

He has had, from time to time, chills and fever, but not so frequently. He was in the county hospital some three years ago for about seventy days, with what was diagnosed by his doctor as bronchitis. In 1879 his doctor told him he would be better off if he left Indiana, for he had that condition known as "ague cake." He has now a tumor in the region of the spleen. The fact that his doctors stated to him ten years ago that he had then an ague cake is proof that the tumor has been there ever since that time. For the last six years he has had more or less yellowness of the skin.



Now, assuming that he had malarial fever eleven years ago, it is not strange that he should have had an enlargement of the spleen; and if a spleen in this disease becomes large, it is very likely to continue large. Indeed, a very large spleen resulting from malarial fever never returns to its normal size; it may become somewhat reduced. And if it is true that he had malarial poison, that the symptoms rapidly manifested themselves, and the doctors assured him he must leave the country because of this condition, it is not surprising that we should find a tumor there now. But it is important that we should ascertain whether the tumor is the spleen, for other tumors are found sometimes which resemble an enlarged spleen in the place where an enlarged spleen is found. It has not increased in size, probably, in the last two or three years. That is the belief of the patient. A little over two months ago he came to me and I examined it; it has not increased in size since then, but is perhaps slightly reduced. It feels hard. The edge is distinct and does not seem to be nodulated. It comes to about the median line, and reaches down below the level of the umbilicus.

What other tumor could occupy the space and position this one does? A tumor of the left kidney would probably not come up to the surface as distinctly as this tumor does. Moreover, it would be more oval or round, less flat, and distinctly edged. A cancer of the left kidney might occur and occupy the left portion of the abdomen. A tumor developing from where the colon makes a turn might fill a considerable space at this point, only the colon does not reach up so far. If the colon, moreover, was diseased to the degree necessary to make a tumor of that size, of any shape, the patient would have difficulty in the action of the bowels. He has none except a diarrhoea sometimes and sometimes a constipation; no pain, tenesmus, or bloody stools. He might have a tumor of the pancreas that would make a large mass in this region. Such tumors are cancerous or cysts, carcinoma or sarcoma. Certainly neither of those tumors of the malignant sort could exist as long as this has, nor occupy just the position of an enlarged spleen; they would be more central. Various tumors develop from the walls of the abdomen, front, sides, and back, that may be as large as this, and that may lead to confusion. The books say very little about this matter, but I am naturally on my guard about the possibility, because I have had one experience of the kind.

A young man had a very large tumor in the exact position of an enormous spleen; it had every appearance of a splenic enlargement, except, perhaps, an indistinctness of the border notch, and its size, which was so colossal as to throw some doubt on that theory. It had been slow of growth; it was uniformly hard, and had a distinct, sharp border. There was no history of pronounced ague. The white corpuscles were moderately increased in proportion to the red. Indigestion came on rather late, with slowly progressing debility. Before death two other similar tumors appeared, one in the lower right-hand portion of the abdominal cavity, the other on the scapula. Post mortem the large tumor was found to be a mixed growth, mostly cartilaginous, with spots of mucoid, sarcomatous, and other developments, and to have sprung from a transverse process of a vertebra. The spleen was of normal size and crowded up against the diaphragm. The small tumors were cartilaginous.

A hard tumor remaining long and producing very little injury to the system is very likely to be a chondroma,—a tumor development from some of the skeletal structures. How can we determine whether this is a chondroma or an enlarged spleen? It might be very difficult, but there are a few points that are of value that may assist us. I think we can rule out all the other tumors, and say that this is either an enlarged spleen or a mixed tumor of the slow-growing kind, mainly chondromatous. We should not be surprised at a slight displacement of the spleen at times as well as enlargements. If it is adherent, it might be adherent to some point that would make the next step in the diagnosis one of uncertain value. That step is this, that a tumor that develops in the spleen or in the colon moves up and down with the movement of the diaphragm. If it develops from the skeletal structures, it is likely to be fixed. But if it becomes firmly adherent superiorly, it might receive some motion from the diaphragm. I put my fingers over this man's enlargement, and feel that it moves up and down with the movement of the diaphragm. That shows that it is not, probably, fixed to the bony tissues posteriorly. This feature points in the direction of a tumor of the spleen. Let us now see if the tumor extends up under the borders of the ribs above, where a spleen ought to be. You know that the normal spleen lies between the ninth and eleventh ribs, and that its dulness should never be perceived forward of a vertical line drawn through the centre of the axilla; its dulness is posterior to

this rather than otherwise. We find by percussion that this tumor does reach up under the ribs. Finally, we find—the best test of all—that there is one irregularity in the anterior border of the tumor,—a depression that is probably the enlarged and somewhat changed notch normal to the edge of the spleen. The doctor in Indiana was doubtless right that it was an ague cake. The patient's temperature two months ago was  $101.5^{\circ}$  F. He had a diarrhœa at that time, had been eating indiscreetly, and I have never found him with more than half a degree of fever since. I examined his blood, and found there was no material change in the proportion between the red and white corpuscles, and I found no pigment to amount to anything, and none in the white corpuscles.

We must remember that spleens become enlarged in various ways. Malarial poison is one cause. That produces the chronically enlarged spleen, the ague cake. And there is something peculiar about this. One man will have repeated attacks of ague, and the spleen will never become markedly enlarged. Another person, right by his side, taking in the same poison from the same locality, will have no worse ague, and yet his spleen will become enormously enlarged and will remain enlarged permanently. We do not know the explanation of this circumstance. Pigmentation occurs when the spleen is enlarged, even when no fever is present. Sometimes it takes place to such a degree that the skin becomes considerably darkened by it. In all cases of malarial fever there is such destruction of the blood as to lead to some pigmentation of the white corpuscles.

This patient is very anxious to be well. As I have said already, we cannot promise him that the spleen will ever return to its normal size. There are two reasons. One is, that, as a matter of fact, these enlargements do not disappear entirely; the other is, that we know that in all these cases the tissue that makes up the major part of the mass of the solid portion of the spleen has increased in size. There is an increase in its substance, and it is like the adventitious tissue of the liver in cirrhosis,—it never disappears wholly. As to what the prognosis should be in a case of this sort, we should consider, first, the natural history of the case, and secondly, the condition of the patient. An enlarged spleen from ague we know may remain enlarged for many years and the patient enjoy fair health. This is so if the spleen can cease to grow larger and be free from tenderness,

and the patient can nourish himself and keep up a fair quality of blood. This man has had no tenderness except at the point where the blood-vessels enter the spleen, and I think we may assume that this tenderness will not increase, and that it ought not to handicap him very much in maintaining his health. It may be due to a perisplenitis, and the organ may be adherent to the abdominal wall to a slight degree. When that condition exists, the movements of the body sometimes cause pain. The general condition of his health otherwise is not good. There is a tendency to a recurrence of fever that menaces this patient always. Any little disturbance of health, as overwork, indigestion, or cold, causes fever, showing that he is laboring both under great debility and some poisoning. The pneumonia he had some years ago, and the bronchitis that kept him in the hospital seventy days, would not probably have occurred except for this enlargement and its consequences. He is not a drinking man, and is careful not to overwork, but the evidence of the jaundice, the discoloration of which is shown most in the whites of his eyes, is to me the most marked indication of a disorder in connection with the liver. In a profound ague, and in a profound seizure of remittent fever, we can always find the liver enlarged. It is always pigmented as the spleen is pigmented. Rarely does it remain enlarged, and this man's liver is not enlarged at all. He may have a chronic duodenal catarrh. When a little child he drank lye, and had difficulty in swallowing until he was fourteen years old. He had to have a probang put down his throat more than a dozen times, he says. I am glad he mentioned this, because it is instructive in connection with a case we had in the lecture-room last Tuesday, of a child whom I advised to go home and do nothing in the way of operative treatment, and that advice was based upon the hope—and the hope was founded upon the experience of the past—that as the child got older and larger the difficulty would decrease. This patient swallowed lye when but two years of age, and a difficulty in swallowing for twelve years afterwards was experienced. The difficulty was not so great as in the child's case referred to. He has gotten over it.

Even with all his drawbacks, I should say there was a fair prospect that, with good care and treatment, this man's tumor may be prevented from growing larger. His health will never be good. What treatment can we give? The first consideration must always

be the hygienic. This patient should be put in as good condition as possible. He is unfortunate that he must earn his living. He must be kept out of malarious atmospheres. I am not certain that the atmosphere of Illinois is the best. He must take an abundance of rest and sleep, and various drugs that have a wholesome effect. A considerable number of the drugs lauded for this disease probably have no value whatever. But moderate doses of quinine are valuable, and small doses of iron sometimes help these patients markedly. The arsenical preparations have been praised very much; before the discovery of quinine that was the great remedy. Moderate quantities are eligible in cases of this sort. The arsenate of sodium is less likely to irritate the stomach than other preparations. It is a preparation very rarely prescribed, I am sorry to say. This patient has had this treatment, and he has improved somewhat. Various local applications have been resorted to in these cases. They are of no particular value, except the mercurial ones. (Patient dismissed.) Now, while I assure you that, from all the considerations we can discover in this man's case, it is one of enlarged spleen,—the so-called ague cake,—and while everything I have said has been entirely frank, and said in his presence, I should be glad to say, out of his presence, that in such cases we should always hold our minds ready to discover that some other disease exists in the spleen or about it than this one. Various changes come on in the spleen, and if this man should die and we should secure an autopsy, and not discover an enlarged spleen, with the trabecular matter everywhere thickened, we might find a neoplasm. The right border has not what the common enlarged spleen shows us of an ideal notch, although it has a notch, such as it is. But in view of all the circumstances, the balance certainly tips in the direction of an enlargement of the spleen from malarial poison. There is another fact worth considering. In former years he drank a great deal of whiskey and other liquors, which doubtless depressed his system and made it easier for any poison to produce injury upon it.

#### FIBROID PHTHISIS.

We have here a patient with the following history. The man is thirty-six years old, and has been sick one year; he has been coughing more or less during that time, and six weeks ago he commenced to be hoarse, and at the same time he had night-sweats, which have

continued constantly since. It is difficult for him to sleep on account of coughing at night. His appetite is not good; he says he has lost a number of pounds in weight. His family history is good; there is no history of any pulmonary disorder. But it goes without saying that when a patient comes into a clinic complaining in this way that the suspicion is at once raised of phthisis, and it is in reference to that point that my assistant has inquired as to the family history. The patient's temperature is just 100° F. at this time. His pulse is 120, but I presume it is somewhat excited by his coming into this room and the examination he has undergone. His voice is hoarse, as you will notice. Now, what would all these facts suggest to your mind?

*Student.*—Phthisis.

*Professor.*—Anything else? That would be the main one, probably, but cannot you think of any other disorder or disease that might produce such symptoms and such a history as that? Severe bronchitis might produce all these symptoms. What else?

*Student.*—Acute pneumonia might.

*Professor.*—Only the trouble with that theory is that the man gives a history of having coughed for a year and a half and has gradually become worse. Were you any worse six months ago than a year ago?

*Patient.*—I got very much worse six weeks ago.

*Professor.*—Did you have to go to bed?

*Patient.*—Yes, about two weeks.

*Professor.*—He says he was in bed two weeks. What do you spit up when you cough?

*Patient.*—Sometimes chunks.

*Professor.*—How do they look?

*Patient.*—A sort of green color.

*Professor.*—What was the color of the material you coughed up prior to six weeks ago?

*Patient.*—Almost white.

*Professor.*—Those were doubtless masses of muco-pus; they come from his bronchial tubes in all probability. If he had had lobar pneumonia six weeks ago he could have recovered from it by this time, and certainly ought now to have assumed his habits of life, but as a matter of fact he is a very sick man. While he was in bed he was sicker than he is now. That he had some acute sickness is

true, but he recovered sufficiently to get up. He has had night-sweats ever since, and has been losing weight, and has been unable to work. If he had pneumonia, it must have been the catarrhal form, in which there is no expectoration of rusty sputa, so characteristic of lobar pneumonia.

Now, there is another fact that has an important bearing on his case, and that is the hoarseness. That came on about the time this sickness occurred, and that is a symptom very likely to occur in cases of tuberculosis. Let us examine him and discover the lesion if possible. It is only by physical exploration that we can settle the question. Now, we will first observe the appearance of his chest. Both sides seem very much alike, possibly there is a little less motion on the right side than on the other. Let us see if we can detect any difference between the two sides in the transmission of vibratory movements or impulses. (The patient counts one, two, three, four.) The fremitus seems to be more pronounced on the right. It can be felt better on the upper portion of the right side, and therefore this part of the chest, lung, or what not has more capacity for transmitting those vibrations of the voice that are perceived by the hands. Let us now by percussion test the capacity of the different parts of the chest as to resonance,—to change the qualities of sound produced by a blow. We percuss first with the patient's mouth closed. We will next ask him to open his mouth wide and breathe through it while we percuss. The pitch may be a little higher on the apex of the right, but the sound is more resonant under the right clavicle than under the left. It is a little higher on the right side than on the left; but how can it be more resonant there and be higher in pitch?

*Student.*—Possibly there is a cavity there.

*Professor.*—I must say that to my ear the percussion characteristics are not sufficient to enable one to say just what condition is probable without listening, interesting as such an inquiry always is. It is true that a sort of tympanitic tone is sometimes produced by a little cavity; a peculiar resonance is produced in that way. Percussion over other portions of the chest reveals normal sounds. Elevation in percussion pitch usually goes along with some quality of dulness, a result of the lessening of the size of the normal air-spaces, and an increase of the quantity of tissue in a given area. Nearness of a cavity or dilated air-vesicle changes the relation of these ele-

ments. Percussion over the right apex with the mouth open fails to produce the cracked-pot sound of a cavity.

Let us now test the capacity of the lungs to transmit and change the sounds produced by movements of air into and out of them, by means of auscultation. We listen to the normal breathing movements, and find that over the right apex the expiratory sound is longer than the inspiratory, and apparently or really higher in pitch. This means that the solid tissue of the part is increased in amount, and its elasticity lessened. Instead of the normal slight puff of low-pitched expiratory sound, we have a sound prolonged by slow collapse of the lung, and made tubular and blowing by passing through tubes reduced in diameter and roughened in their lining, while its pitch is elevated by being transmitted through solid tissue,—more solid tissue than is normal. I cannot hear a true cavernous sound over the right apex, as I might if a cavity of some size were near the surface of the organ. Next we will test, by auscultation, the capacity of different parts of the lungs to transmit the vibrations of the patient's voice. Over the right apex the voice and whisper are heard with slightly more distinctness and loudness than on the corresponding region of the other lung. This is further evidence of consolidation; increase of the lung tissue at the expense of the air-spaces.

The thing that does not seem to be explained so far is the slight evidence of increased resonance on the right side in front. What can explain it? I know of no explanation except this, that, as suggested, a cavity exists in the neighborhood, or that perhaps, and probably, affected portions of the lungs have undergone consolidation, or an increase of connective tissue that, by contraction and cutting off of blood-supply to certain septa between air-vesicles, has caused their absorption and the reduction of the number with increase of the size of the spaces, probably near the surface. So that we have to a degree that condition which is known as fibroid phthisis. It is rarely equally distributed through a lung. Now, there is no way probably of our settling the question absolutely as to whether this man has tuberculosis except by examining his expectoration. But there is one other confirmatory evidence that we may reach to-day. There is nearly always a swelling that is somewhat characteristic in cases of tuberculous laryngitis. The swelling is usually above the cords and back of them. I mean by this to refer to a condition prior to ulceration. At certain points, especially over the



arytenoids, the mucous membrane often appears swollen, indicating the seat of deposit where ulceration may be looked for. But other parts of the larynx may first be involved, or the epiglottis. The swollen region is not always red, but has sometimes a grayish, translucent appearance. Later on it becomes red, and so we may find it in either condition. If in a patient with such a history as this we find this tissue swelling in the manner referred to, it is almost positively diagnostic of tuberculosis. Now, on looking into this man's larynx with a laryngoscope, I should say, from what I can see, that there is no great enlargement of the arytenoid region; there seems to be a rough spot on the left vocal cord near its base, probably a tuberculous ulcer. So that all the points we can get from this patient seem to point almost conclusively to the presence of tuberculosis of the apex of the right lung and of the larynx. The expectoration has not been examined, but must be. If several successive specimens of his expectoration reveal no bacilli, that would negative our suspicions of tuberculosis. If we had not examined the patient at all, but found his expectorations to swarm with bacilli, that would settle the question.

If the patient has tuberculosis, there is small chance of his recovery in this climate. If he had friends who could send him to some genial climate, he should be sent immediately. He ought to be sent South, but not to a high altitude. Some of our Southern States would be better than Colorado. He should have, in addition, all those aids in the way of medicine, regimen, food, and conditions that will increase his vigor,—that will improve his natural physical capacity to resist tuberculosis. For we must not forget that this is the great remedy—if there is a great remedy—for this disease. No remedy can be of much consequence in the absence of a good capacity of blood-making and of resistance.

#### ENDOCARDITIS.

This patient is a young girl who has been sick since July last. The first thing she noticed was what she calls a tired feeling, later a pain; the pain seems to be on the left side of the chest, where there is a slight tenderness, and it hurts her to take a deep breath. She says she is short of breath, a symptom she first discovered about six months ago. She says she did not catch cold nor have a chill at that time, and that she did not lose her appetite. She had sensations of discomfort over the heart, and as a result of this sensation, as she thinks, she would get very weak at times and would faint away. She

did not know or have a suspicion of what was actually the matter with her. She thought she had just tired herself out and was overworked. She has been short of breath since that time, and has lost her appetite. She sleeps well enough, but for about three months she has had a cough. She says she coughs when she walks up the stairs; exercise makes her tired and gives her a sense of breathlessness. There is no dulness on percussion over the chest, except possibly a slight increase of the cardiac dulness. The heart's apex is to the left of its normal site, and is to the left because the organ is enlarged. It is therefore probably a case of endocarditis, with consequent cardiac hypertrophy. This is a very interesting case, because it illustrates how it comes to people who have supposed themselves previously well. This patient says that she felt as though she was tired out and overworked. She was sent away, and improved, but this breathlessness came on, and she has never been up to par since. In some stupid people this occurs without their observing it. Nothing especially pointed to the heart but these pains, which were probably not in the heart at all, though possibly some of them were. Many times, especially in children, endocarditis occurs, and the symptoms are such as when a child has caught cold or is a little bilious, and many of those cases are never seen by a physician at the time of the acute sickness, never until the short-windedness comes on later, and then they are taken to a doctor to see what is the matter.

On listening over the heart we find just beneath the apex-beat, to the right and to the left of it, a systolic blowing sound that is slightly musical. The musical quality is unusual to heart murmurs, and is not satisfactorily explained. It is not heard at the base. Down low under the apex it is not heard, or it is rather faint. It is heard also far to the left of the heart and in the left interscapular region. It is not so very important just where the lesion is, but it is important whether it is at a stand-still or progressing, and whether the heart is greatly handicapped by it. This is evidently a case of endocarditis with mitral regurgitation.

Good management is required in a case of this kind, and that management consists in all those things that will enable the patient to exist and go on living with the least pain and discomfort. She should be spared excitement of body and mind that can overwork the heart, and should live a tranquil and physiological life, that will minimize the danger of a recurrence of the endocarditis.

# ULCERATION OF THE PYLORUS AND ITS CONSEQUENCES; DILATATION OF THE STOMACH, WITH REMARKS AS TO THE TREATMENT.

CLINICAL LECTURE DELIVERED IN THE ROYAL INFIRMARY.

BY THOMAS OLIVER, M.A., M.D., F.R.C.P.,

Physician to the Royal Infirmary, Newcastle-upon-Tyne, England.

---

GENTLEMEN,—In the North of England, chloro-anæmia is pretty prevalent, and one of its consequences—ulceration of the stomach—is frequently met with in young women. At a more advanced age of womanhood, and as a sequel of ulceration of the pylorus, there is often dilatation of the stomach. The female sex is the more liable to suffer, but men are by no means exempted.

In Allbutt's "System of Medicine," vol. iii. p. 519, it is stated on the authority of Bamberger that ulceration of the stomach is oftenest observed in cooks, a circumstance attributed by him to their habit of tasting hot food; but my own experience leads me to regard housemaids and general servants as being even more liable to this illness than cooks. Hot food, therefore, cannot be the cause of ulceration of the stomach. Chloro-anæmia and amenorrhœa seem to stand to it in a closer though more obscure causal relationship. I have seen little of tubercle as a cause, and so far as syphilis is concerned, while it is known to create a tendency to the development of endarteritis and thrombosis, yet these lesions are seldom met with in the stomach. When syphilis can be blamed, it is probably through the diseased conditions established in the submucosa, a circumstance which explains the greater destruction of the mucous membrane then observed, also the irregular borders and the absence of the punched-out appearance of the ordinary peptic ulcer.

Ulceration occurs near the pylorus and on the posterior wall more frequently than at other parts of the stomach. While these are the more usual situations, the greater curvature is not exempted, but

there is this very important difference. When ulceration occurs at the cardiac end of the organ, there is a greater tendency for healing to take place than when it is near the pylorus, the reason being that there is less movement at the proximal than at the distal end of the stomach; that the mucous membrane is less bound down and therefore less likely to be stretched. As a rule, there is only one ulcer present, but there may be more. Multiple ulcers are much more likely to be the result of bacterial infection than a solitary ulcer. Round, with clean edges, and of varying depth, ulcers of the stomach present a peculiar funnel-shaped appearance which is more or less characteristic. The breach of surface in the gastric mucous membrane may be recovered from, a puckered cicatrix subsequently marking the site of the ulcer. Perforation may take place, causing rapid death, or this latter is prevented by adhesions forming. Occasionally the inflammatory material that is thrown out is absorbed, and, as happened in one of my own cases, the ulceration extended into the head of the pancreas and caused death by eroding an artery. When perforation occurs the opening is usually not very large, a fortunate circumstance for the patient, since as only a small quantity of the contents of the stomach escapes, peritonitis with adhesion is induced, and for the time being, at any rate, death is averted. But the opening may be larger, as, for example, that which occurred in a young girl who had no symptoms beyond slight indigestion, and who was not under treatment. Seized with pain in the abdomen and an inclination to vomit, she suddenly gave a cry as if in intense pain, fell down in a state of collapse, and died in a few minutes. In her stomach I found an ulcer the size of a shilling, with a perforation in its floor through which I could readily pass my finger.

The causation of ulcer of the stomach is obscure. Probably in most of the cases there is more than one cause in operation. In unhealthy conditions of the blood, pathological changes in the walls of blood-vessels, abnormal states of limited areas of the mucous membrane, and in altered gastric secretions lie most of the causes of ulceration of the stomach. The altered blood condition most frequently found associated with ulceration of the stomach is chloro-anæmia, and just to the extent that living in imperfectly ventilated rooms and eating unsuitable food predispose to chloro-anæmia, so may these be regarded as causes of ulceration. But as ulceration of the stomach is by no means confined to people who are poor, or

who are engaged in domestic service, so chloro-anæmia must of itself, in some way or other, lead to the illness. Of course, many of the girls suffer from amenorrhœa also, and while this may be a consequence of the chloro-anæmia, yet as amenorrhœa is generally accompanied by obstinate constipation, the two things together render the blood still more toxæmic and increase its deleterious influence. In anæmia there is not only malnutrition of the walls of the blood-vessels, but a tendency for the white corpuscles to adhere to the interior of the small vessels and induce thrombosis. Should this occur in some of the arteriocapillaries of the stomach, ulceration would inevitably follow. It is maintained that excessive acidity of the gastric juice may be a cause of ulceration, but in a healthy stomach, with normal blood coursing through its vessels, the influence of this can only be slight and transitory, except in cases where food has been retained in the organ for a length of time, or thrombosis has occurred. I have no experience of traumatism as a cause, and although loss of nerve-power is a likely cause, it is difficult to prove the relationship. To Dr. Soltan Fenwick we are indebted for the opinion that the establishment of inflammatory lesions in the lymphoid tissue, which is here and there massed into heaps under the gastric mucous membranes, is a common cause of ulceration. Inoculation of the gastric mucous membrane with pure cultures of typhoid bacilli has produced the lesion in question. In the experiments performed by Boetcher and Letulle these inoculations were followed by hemorrhagic erosions, which, had time been given, would probably have ended in ulceration. If chloro-anæmia is alone capable of causing this stomach affection, so does the altered blood of pregnancy. I have seen two or three deaths occur from hæmatemesis a few days or weeks after parturition, and where everything pointed to the fatal termination being due to ulceration of the stomach. Generally there has been in these patients pretty profuse hemorrhage at the time of the confinement, and this has been followed by marked anæmia. This clinical fact lends weight to the views put forward by Quincke and Daettuyler (Allbutt's "System of Medicine," vol. iii. p. 526), who, in their experiments upon dogs, found that when these animals were rendered quickly anæmic by bleeding, they suffered from ulceration of the stomach after irritation of its mucous membrane, and that the lesions failed to heal.

In this lecture I am less disposed to deal with the symptoms

of ulceration of the stomach and duodenum than with its consequences. Briefly, however, the symptoms are pain after eating, relieved by vomiting; pain referred to the epigastrium and spine; hæmatemesis, and mælæna. In many patients, pain and vomiting are absent: without premonitory symptoms, they suddenly bring up mouthfuls of blood. Ulceration may even proceed to perforation without pre-existing symptoms.

It is to dilatation of the stomach as a consequence of ulceration at or just beyond the pylorus that I wish to draw attention in the following three cases:

1. *Dilatation of the Stomach; Ulceration of the Pylorus; a Lengthened History of Pain and Vomiting; Operation; Recovery.*—Isabella P., aged thirty-nine years; a widow; admitted complaining of pain at stomach of four years' duration, and of having vomited for the last three years. Family history good. Since the death of her husband she has worked as a charwoman in order to support herself and her four children; has had a good home and plenty of food. Four years ago she began to experience an aching pain in the stomach extending to the spine, particularly after eating. A year after this, vomiting came on and has been repeated every two or three days since, patient frequently vomiting a quart of liquid at a time, sour tasting and foul smelling. Patient is pale and thin; she has been emaciating, for she is now only five stones one pound (or seventy-one pounds) in weight, having lost fully twenty-eight pounds within the last two years. A tumor can be felt below the right costal arch in the situation of the pylorus, and splashing can be both heard and felt when the upper part of the abdomen is palpated. An area which is dull on percussion and somewhat curved, commencing two inches above the umbilicus and extending into the lower part of the abdomen, can be mapped out, but it is noticed that the dulness alters with any change of position of the patient. Into the stomach, by means of a syphon tube, six pints of warm water were carried, and the whole of the six pints were again removed. This is nearly double the capacity of most human stomachs. The heart and lungs are healthy, and the urine also. Treatment by means of salicylate of bismuth and lavage were tried, but as the patient did not put on weight or her pains lessen, I asked my surgical colleague, Mr. Williamson, to open the abdomen and explore the neighborhood of the pylorus. He did so, and found, on

slitting open the pylorus, a large, deep ulcer, the size of a florin, the edges of which were soft and sloughing. After clearing these, he brought the edges of the wound made into the pylorus into apposition by suture, the line of the incision being brought so as to lie at right angles to the lumen of the gut. A Lembert's suture of fine silk was then carried through the serous coat of the stomach, and the abdominal wound closed by means of ordinary silkworm-gut. The patient made an excellent recovery and remains quite well.

2. *History of Recurrent Epigastric Pain and Vomiting; Symptoms of Dilated Stomach; Operation; Death Three Days after from Hemorrhage, the Duodenal Ulceration having extended into the Pancreas.*—Annie McC., aged fifty-six years; a widow; admitted complaining of pain in the stomach and vomiting of nineteen weeks' duration. Family and personal history good. Six years ago she had the menopause; she is the mother of five children. Nineteen weeks ago she began to have attacks of severe pain over the stomach without any apparent cause. They were not always aggravated by taking food. Sometimes they would last all day; they were never followed by jaundice. Occasionally a hard swelling could be felt in the upper part of right abdomen. Patient vomited once or twice every day, the fluid expelled being frequently bitter to the taste, pale in color, and inclined to ferment on standing. The vomit never contained blood. It varied in quantity, occasionally as much as a quart being brought up. Her internal organs, other than those with which we are particularly concerned, were healthy. My diagnosis was dilated stomach, probably due to a stenosed pylorus, and, as the patient was past the middle term of life, there was just the suspicion that there might be malignant disease. Pain was so severe, and it had latterly become so constant, that she begged for the abdomen to be explored. Mr. Williamson, at my request, opened the abdomen by a vertical incision over the pyloric region, and, drawing the pylorus forward through the wound, he found its walls thickened and that the thickening extended well into the duodenum, which was adherent to the gall-bladder. An incision was made through the pylorus and first part of the duodenum in the long axis of the gut, and the parts subsequently sutured in accordance with the methods usually adopted in such cases. Patient was much collapsed after the operation, but she rallied in the course of the day. Next day she vomited a large quantity of bright-red blood, and again

seemed very collapsed. On the following day she died, and at the autopsy we found the peritoneal cavity quite clear and healthy, the stomach slightly dilated and containing fully a pint of blood, its walls thin, and its mucous membrane healthy as far as the pylorus. Immediately beyond the pylorus, on the posterior wall of the duodenum, was a large irregular circular ulcer, the size of half a crown; its edges were undermined. On raising the undermined edges the ulcer was seen to have formed a considerable sized cavity, smooth-walled, in the head of the pancreas. Stretching across the floor of this was a large blood-vessel pretty fully exposed, a radicle of the pancreaticoduodenal artery, the wall of which had become so eroded that it could readily admit a director. From this artery the bleeding had occurred which caused death.

3. *History of Old-Standing Dyspepsia, of Recurrent Vomiting, and "Water-Brash" of Ten Years' Duration; Signs of Dilated Stomach due to Pyloric Stenosis; Operation; Recovery.*—Michael H., aged fifty-three years; a steel grinder; admitted complaining of indigestion of seventeen years' duration, of pain over the stomach and water-brash of ten years' duration, and of vomiting, especially in the evening. Ten months ago while at work he became rather more violently sick than usual, and since then he has vomited almost daily. He has lost five stones (seventy pounds) in weight. Of an evening he would frequently vomit two or three pints of a foul-smelling liquid, which if allowed to stand over night appeared like yeast fermenting. His internal organs, generally speaking, are healthy. Splashing can be readily elicited by tapping the upper half of the left rectus abdominis, while waves of muscular contraction can be seen running slowly from left to right. On passing five and a half pints of warm water into the stomach the outline of the viscus could be readily observed, extending from two inches above the umbilicus to two inches above the pubis. In the vomit *sarcinæ ventriculi* were found. As the case seemed a suitable one for operation, Mr. Williamson made the usual incision into the abdomen, and found the pylorus adherent to the gall-bladder. These adhesions were divided and the pylorus brought forward and incised. The pyloric orifice was found to be obstructed by dense cicatricial tissue, so the incision was carried right through the orifice for some distance on either side. The central portions of the incision were drawn out laterally and sutured, and the operation concluded in the usual manner. The patient made an excellent recovery.



So far we have regarded dilatation of the stomach as the result of simple ulceration at, or just beyond, the pylorus. There are physicians who maintained that a functional obstacle like pyloric spasm is capable of causing dilatation, and that the spasm is due to a hypersecretion of the hydrochloric acid of the gastric juice. That an excess of this acid in the stomach is capable of inducing pyloric spasm has been demonstrated by Ewald and Boas, von Pfungen, and Uleman, who found that an average degree of acidity of one and a half per thousand excited peristaltic action and promoted emptying of the stomach, whereas a higher degree of acidity—*e.g.*, two and a half per thousand—brought on spasm of the pylorus and interfered with the onward passage of the food into the intestine. Hyperacidity is therefore capable of inducing spasm of the pylorus and thereby of causing retention of food in the stomach, and one of the consequences of this retention is that the amount of acidity tends to be still further increased. Long-continued and repeated spasm would be ultimately followed by fibrous thickening of the pylorus; hence cases of stenosis of the pylorus with thickening of the walls without ulceration. Professor Carle, of Turin, in the course of eighty-four operations on the stomach in the Ospedale Mauriziano of Turin (*Medical Weekly*, August 6, 1897, p. 361), found nine cases where the food had been retained in the stomach owing to permanent spasm of the pylorus. While hyperacidity of the stomach's contents and pyloric spasm act and react upon each other, it is interesting to know that once the stenosis is overcome, as by operation providing a ready escape for the food, this intense hyperacidity disappears. It is of course quite possible that surgeons, in their operations upon the pylorus, may have overlooked the existence of very small ulcers or of their cicatricial remains, but it is none the less true that this part of the stomach has been found thickened, or an accompanying peripyloritis has existed without any trace of ulceration.

Pyloric obstruction is the chief cause of dilatation of the stomach. The lesion may be internal, as in ulceration, or external, as in adhesion or the presence of a growth. Retention of food in the stomach owing to muscular atony is also regarded as a cause, but in such, whether pyloric spasm may have existed or not, the lengthened delay of food, owing to enfeebled motor activities and deficient digestive powers of the gastric juice, is certainly likely to favor dila-

tation. There is a form of dilatation of the stomach alluded to by Allbutt, and regarded as the result of toxæmia, occurring in the course of fever, pneumonia, and influenza, which is not without danger. It is rapid in its development, it retards convalescence, and if not treated it is occasionally fatal. Probably in these cases pyloric spasm is present. Since atony of the muscular wall of the stomach alone will not explain the dilatation, it is thought to be the result of the operation of a toxin or toxins upon the pneumogastric nerve, which, becoming paralyzed, there follow feebleness of the walls of the viscus and a deficiency in the secretion of the gastric juice. Gastric catarrh is also regarded as a cause of dilatation, but it is difficult to say how it acts, unless through a weakening of the digestive power of the gastric juice, which in its turn would favor fermentation and allow of the food lingering in the stomach. All forms of obstructive disease at the pylorus, however, are not followed by dilatation of the stomach. In several cases of cancer of the stomach that have come under my observation, with marked pyloric thickening, the organ has retracted and been sometimes only half its normal size. The obstruction was just as extreme, but, in consequence of the long-continued cachexia and the loss of appetite, only small quantities of food would be passed into the stomach, there would be little functional activity, and so the organ would become atrophied. Something more than pyloric obstruction is therefore required to cause dilatation of the stomach. In fermentation of the food, hyperacidity, abnormal exhalations from the mucous membrane, in pathological changes induced in the nerve-twigs and muscle-fibres of the wall of the stomach, will be found conditions which, co-operating with pyloric obstruction, produce the diseased condition in question.

*Symptomatology of Dilated Stomach.*—Sometimes dilatation of the stomach reveals itself by no symptoms other than those of flatulent dyspepsia; at other times patients look pale and sallow, seem feeble and are emaciated, have a long drawn face with deep grooves encircling the corners of the mouth, their breath is sour, their skin dry and rather cold, their bowels are confined, and their urine is scanty. There may be complaint of abdominal pain and vomiting. Vomiting usually occurs at night, after several meals have been taken, of large quantities of sour-smelling liquid, which, when allowed to stand over night, undergoes fermentation, dividing itself into three layers,—the lowermost composed of grayish-brown *débris*,

the middle layer of greenish liquid, while the uppermost is a frothy foam in the depths of which are found yeast-cells and *sarcinæ ventriculi* on microscopical examination. An individual thus afflicted is nervous and depressed in spirits; he is unhappy and miserable in his surroundings; he is unfitted for exertion, and his sleep is broken. In addition to being emaciated, his abdomen is retracted; its walls may be so thin that the outline of the stomach can be observed, and passing over it may be seen and felt waves of peristaltic contraction. There is dulness on percussion over the lower half of the stomach area, and this is observed to be carried downward and further to the right than normally. The peculiarity of the dulness of a dilated stomach is that it alters with the position of the patient. Marked off by a pencil when the patient is in the upright position, the dulness will be observed to shift as the individual lies on his back or on his side. Add to these physical signs the splashing sound which can be generally heard and felt when the abdomen is gently struck, and we have a pretty complete symptomatology of dilated stomach. It may be necessary to test the capacity of the stomach, either by passing a measured quantity of warm water into it, when anything above three pints will suggest the probability of dilatation, or the organ may be inflated by carbonic acid gas, such as will be evolved in making the patient drink the two parts of a *seidlitz* powder separately. I have known this capacity test fail. It deceived me in one patient whose stomach readily received five and a half pints of warm water, most of which was returned, and yet the stomach was not found to be dilated, and the same remark applies both to the splashing sound, the presence of *sarcinæ* in the vomit, and the fact of the vomit fermenting: these were all present, and yet at the operation there was no dilatation of the stomach. A more reliable test is the length of time the food remains in the stomach. It is astonishing how long food will remain therein undigested. In one of my patients,—an old coal miner,—when we washed out his stomach for the first time we removed a large quantity of currants, which seemed as fresh as the day on which they had been eaten, and yet they had lain in the stomach for upward of a fortnight. If the test of inflating the stomach by gas is to be tried, the organ should always be washed out first, otherwise the extremely acid character of the contents will prevent the evolution of the gas.

Given a patient presenting symptoms of dilated stomach pre-

sumably due to ulceration at the pylorus, it is of extreme importance to know whether the lesion is malignant or non-malignant. In dilated stomach caused by simple pyloric obstruction the vomit is intensely acid; not only is the hydrochloric acid increased, but other organic acids are present,—*c.g.*, lactic and butyric, due to the decomposition of carbohydrates. Simon ("Clinical Diagnosis," p. 136) states that only in one disease—*viz.*, cancer of the stomach—is lactic acid found in notable quantities, and he quotes the facts observed by Koch in one patient who was suffering from ulcer of the stomach and in whose vomit free hydrochloric acid was found. Suddenly the hydrochloric reaction could no longer be obtained. Lactic acid, instead, appeared and increased steadily in amount from week to week, when a tumor began to be felt in the epigastrium. At the autopsy a carcinoma of the stomach was found at the base of a pyloric ulcer. It is now several years since the attention of the profession was drawn to the statement that in cancer of the stomach hydrochloric acid disappears from the gastric juice, and while in many cases this is undoubtedly true and the circumstance becomes of value from a diagnostic point of view, it is not absolutely true. Some time ago I invited our professor of chemistry, Dr. Bedson, to examine the vomit of several of my patients who were suffering from various diseases of the stomach, and he found hydrochloric acid frequently absent in non-malignant disease, and often present, contrary to what we had been led to expect, in cancer.

The treatment of dilatation of the stomach from simple pyloric obstruction is dietetic, medicinal, and operative. Into the dietetic I need hardly enter beyond alluding to the necessity for giving patients light, nutritious food, proteids rather than carbohydrates, since these tend by fermenting to add to the troubles and discomfort of the patient. As for the medicinal, washing out the stomach with boracic acid or Condry's fluid and then feeding the patient is a line of treatment which, when the repugnance to it is once overcome, is always palliative: it makes the individual more comfortable; he begins to look more cheerful, and to put on flesh. In not a few cases of dilatation of the stomach that have come under my care the symptoms have entirely disappeared under this line of treatment, but it has to be carried out for several months. Since in these patients a complete recovery was effected, the probability is that the pyloric obstruction was due to spasm with hyperacidity rather than

to organic thickening, for no amount of lavage could diminish a growth, although by the contents of the stomach being rendered less acid, and therefore less irritating, further increase of the fibrous thickening might be checked.

The treatment of dilated stomach due to non-malignant pyloric obstruction is principally surgical. In my opinion and experience no line of treatment can equal a successful pyloroplasty, either for the immediate and apparently lasting relief which it gives, or for the rapidity with which the appetite is restored, weight gained, and subsidence of hyperacidity secured. I have said "apparently lasting relief," for perhaps the operation has scarcely been long enough in existence for us to speak of a permanent cure, and yet I know of no instances on record in this country where the operation has had again to be performed. The question as to whether in dilated stomach, from pyloric obstruction, pyloroplasty or gastro-enterostomy should be performed is rather surgical than medical. Surgeons are divided as to the respective merits of the two operations. Doyen, for example, is in favor of gastro-enterostomy, and Mikulicz of pyloroplasty. Carle and Fantino, of Turin, base their opinion upon an experience gained out of forty-one operations, fourteen of which were pyloroplasty, of which one died, and twenty-four of gastro-entero-anastomosis, with two failures. So far as the death-rate is concerned, there is little to choose between the two operations. Carle and Fantino are in favor of what is known as postero-gastro-enterostomy, since by this method there is less chance of subsequent adhesion with the omentum and the formation of a spur between the folds. Attention is drawn by them to one sequel of gastro-enterostomy, and that is the tendency for bile to regurgitate into the stomach; but as this circumstance neither stands in the way of a cure, nor prevents the patient subsequently digesting his food properly, they attach little importance to it, since the stomach empties itself of its contents quickly, and, as the organ gradually undergoes diminution in size, a new sphincter is formed which regulates the evacuation of the stomach. After a pyloroplasty, on the other hand, there is no regurgitation of bile into the stomach, the motor functions of the organ are at once restored, so that when recovery takes place the stomach quickly and regularly empties itself, and, although this act is effected less rapidly and the viscus does not become so quickly reduced in size, both are in time as effec-

tively accomplished as in gastro-enterostomy. For cases where there is considerable atony of the gastric musculature some surgeons prefer gastro-enterostomy, but the decision can only be made when at the operation the stomach has been exposed. Circumstances may then suggest to the operator the advisability of his performing gastro-enterostomy to pyloroplasty,—*e.g.*, if the pylorus is firmly embedded in adhesions. Where, too, there is fixation of the pylorus and duodenal ulceration, gastro-enterostomy, by relieving the extrapyloric region of the irritation caused by the passage of food over it, and the more rapid emptying of the stomach, is the safer operation. Both operations are, comparatively speaking, safe. Nearly all my patients upon whom pyloroplasty has been performed have done well and have remained well, but whether in any case it shall be one operation or the other the surgeon can only decide after the abdomen is opened and he has ascertained the pathological conditions that are present in and around the pylorus.

## ANEURISM OF THE ABDOMINAL AORTA.

CLINICAL LECTURE DELIVERED BEFORE THE CLASS OF MARION-SIMS COLLEGE OF  
MEDICINE AT CITY HOSPITAL, ST. LOUIS.

BY I. N. LOVE, M.D.,

Professor of Clinical Medicine and Diseases of Children in Marion-Sims College of  
Medicine, and Professor of the Theory and Practice of Medicine in the  
Woman's Medical College, St. Louis.

---

GENTLEMEN,—We may say that every individual starts out in life with a system of circulation tubes (arteries) that are either in good form or weak. Of course, in the majority of instances the tubular equipment of the body for the carrying of the blood to the various parts of the body is good. There are individuals, however, where there seems to be poor material used, but these are very exceptional. As you know, an artery has three coats,—the internal serous coat, or tunica intima; the middle coat, which is the circular coat and is muscular; the external coat, which is composed of longitudinal, fibrous, elastic, and connective tissue. Another name for it is the tunica adventitia; that is, it is put on for supporting purposes; it is an adventitious coat. Now, the structures of the arteries suggest that we should have proper elasticity; we should have also a proper amount of muscular force, with a view to carry on and assist in the circulation, and with a view to proper contraction in case of an interruption in the calibre of the artery. The internal serous, or epithelial, coat is a smooth coat, which facilitates a ready flow of blood. Every normal individual should have an elastic system of tubing. In the earlier years of life, to middle life, along down the sunny, afternoon side of life, there should be this elasticity, but as age advances there develops what is known as arterial sclerosis, a hardening of the arteries, a lessening of the elasticity. This sclerotic condition develops on the inner coat. It may exceptionally develop in the earlier years of life. Some one has said—indeed it has been an axiom for many, many years—that a man is just as

old as his arteries. In the well-equipped man the artery, when felt, should feel elastic, not firm, not like a goose-quill under the thumb, but soft and pliable; and when you press upon the radial artery you should not be able to feel the artery at all beneath your thumb or your finger at the peripheral extremity of that vessel. If you do, it is suggestive of a lack of elasticity, of arterial sclerosis.

Now, the beginning of this condition, whatever the cause, is a softening, which process may be in certain spots or it may be diffuse, more frequently in spots, and there may develop within that inner coat in these spots a softening almost suggestive of ulceration. Indeed, when the *débris* of this particular spot is noted it looks like pus. Nature very soon throws out or permits to accumulate at these particular points of erosion a deposit known as an atheromatous deposit, and that is conservative, that is nature's means of covering up that weak spot and enabling the artery to maintain its continuity and perform its duty. This conservative effort of nature in throwing out this hard, almost calcareous matter renders the arteries less elastic, it is true, but the continuity of the artery and the circulation is maintained; the individuals may live along for a number of years, particularly if they pursue the proper course of life. When the time comes for them to be taken off, it is very liable to be through the breaking of some one of those vessels, frequently the break occurs in the brain, resulting in apoplexy. That is the way that the old person is frequently taken off.

Now, what is the cause of this arterial sclerosis? I say that it primarily may have been caused by some innate, inherent, inherited tendency or weakness in that direction, but habits of life have very much to do with it,—the excessive use of alcohol, but more particularly excessive eating and interference with the proper metabolism of tissue, by which poisonous matters are retained, causing the uric acid diathesis, the gouty condition. All these conditions are brought about by habits of gluttony, dissipation; and again it may come from mycotic origin; that is, infectious origin, the result of infectious diseases, such as the exanthema. Sometimes we have what is known as malignant endarteritis, a rapid ulcerating inflammation of the lining membrane of the artery. That may be brought about by an infectious disease. Rheumatism is one of the infectious diseases that has produced this condition. Another one is syphilis. That is a very frequent cause of primary arterial sclerosis or athero-



matous degeneration of the arteries. If, during this period of softening,—this so-called ulcerative period of the lining membrane of the artery, before nature in her conservative way has thrown out the plastic materials, enabling the vessel to retain its continuity,—there should be too great a pressure, superinduced by the habits of life, largely by over-exertion, upon the blood column, we may have a breaking of the continuity of that vessel, and a true aneurism be the result, which means the breaking through of the internal coat and the distention and stretching outward of the other coats. Under the head of true aneurism we may have a fusiform aneurism; that is, a spindle-shaped aneurism, a gradual, diffuse dilatation of the outer coats of that vessel. Or we may have a circumscribed aneurism, a pronounced tumor. A false or dissecting aneurism may be produced; that is, the blood will break through the inner coat and dissect around through the other coats, and not produce any pronounced tumor, and then come back at another point into the circulation. Then we may have as the result an aneurismal varix; that is, the breaking of the vessel at this weak point of the artery and a communication between it and the vein. Then you would have a swelling of the vein, a general pulsation and disturbance there, a tortuous condition developed. You would have an accumulation of largely distended veins, the blood again returning, after mingling with the venous blood, into the main circulation.

We have before us William Connell, aged fifty-four. He is approaching the age when there would naturally be developed, if at all, this brittle, diseased condition of the arteries. He is a laboring man. If there be developed within him a diseased condition of the artery, being a laboring man, he is much more liable to the development of an aneurism. We elicit from him the fact that he was admitted into the hospital January 29, about a week ago. His family history is good as far as he knows. He has no knowledge of any hereditary taint. We elicit no further previous history. His habits were alcoholic; has drunk whiskey regularly for many years, often to excess. This fact in itself might be an exciting cause, by a demoralization of the tissues of the body and the resulting arterial sclerosis, which would tend in the direction of developing an aneurism. He had a chancre on his penis twenty-two years ago, followed by suppurating bubo. Of course, there is a doubt here as to whether that was a chancre. He says that he has had eruptions of the skin,

but gives no history of distinct secondary symptoms. He has had pain in his legs lasting a day or two. No history of periostitis, rheumatism, or any acute infectious disease. He was in this hospital from November 12 to November 24. We would state that a diagnosis of aneurism of the abdominal aorta was made then, and his case gave practically the same history and the same subjective and objective symptoms at that time as at present. He has no œdema. The present trouble began about six months ago with pulsation over the præcordia, difficulty of breathing on slight exertion, and a constant dull pain on the left side posteriorly. He lost all desire for food, lost weight and strength, and felt generally miserable. He has had some little difficulty in swallowing solid food, but no regurgitation. The pain, dyspnœa, and cardiac pulsation have grown gradually worse. He locates the pain about the twelfth rib posteriorly on the left side. No vomiting until the past month or so; since then he has often felt nauseated and vomited occasionally. No pain in the abdomen; bowels regular. Entered the hospital with a pulse of 80, respiration 20, temperature 98.

You have already noted by inspection the pulsation in the hypochondriac region. You will also note by inspection a pronounced pulsation in the left side close underneath the lip of the nipple. On putting the hand at that point and at the point below you feel the pulsation, much more pronounced just beneath the nipple than in the hypochondriac region. You notice further that his respiration is rapid and not deep. Oftentimes the pulsation about the heart is very greatly increased; that is, the action of the heart is tumultuous, due to the interference with the circulation beyond. This of itself may gradually be relieved by a compensatory pressure. An aneurism of the aorta may affect the descending, the transverse, the ascending, or the abdominal aorta. If it be the ascending aorta, the symptoms are of course mostly thoracic. There is pressure, and there may be found a pronounced pulsation directly beneath the sternum, and it can be heard also to good advantage at that point. If it affect the descending aorta, it is more apt to be underneath the left scapular region. You can there feel the pulsation and the changed sound, and hear it as well.

Now, as regards the presence of an aneurism of the aorta: oftentimes the most serious aneurism may be present without any physical sign, but with an enormous number of symptoms,—discomforts,

VOL. I. Ser. 8.—11

pain, difficulty of breathing, disturbed circulation, coughing, and all the other disturbing symptoms possible to result from mechanical pressure in the thorax. You may have presented nothing but physical signs, without any previous history or symptom at all. Indeed, the most careful investigation along these lines is necessary. I suggested to you the examination of the radial artery, the pressure, the closing of it. If you feel the vessel distinctly beyond the finger, you have reason to suspect at once an atheromatous disturbance of the arteries, and we have it here. You must take into consideration the age, and whenever you have presented to you a patient with thoracic or abdominal symptoms that are obscure, of course in the primary stages you want to make your investigation thorough. In this case the diagnosis is easy. The exhibition of antisyphilitic treatment, giving the patient the benefit of the doubt (mercury and iodide of potassium), is certainly indicated, and the regulation of the diet so as not to have too great a pressure upon the circulation, the absolute abstinence from alcohol and all stimulants, which increase the pressure, are all of absolute importance. Guarding one's self against exposure to chilling of the surface, against internal congestions, which demoralize the circulation, the guarding against infectious diseases, which of course would aggravate the condition,—all these points are important. A bland, easily digested diet, and then regular habits of life, no violent exercise,—with a proper attention to these rules and regulations the individual who has a pronounced atheromatous disease may escape aneurism, and even though aneurism be present, the patient who properly regulates his life may live many years. You want to take into consideration that if the aneurism is located at the extremity, the popliteal space, for instance, as jumpers and others frequently have aneurisms at that point, it is sometimes cured by pressure, regulating and slowing the flow of blood through the ruptured artery; and by gradual layers a clot is formed, which plugs up the broken artery. I saw a case of popliteal aneurism in which, by constant pressure with the fingers by relays of medical students upon the vessel above, thus regulating distinctly and definitely the flow of blood, slowing it very greatly, the broken vessel was cured. I believe the great Chambers, the eminent medical authority, had several aneurisms which were thus cured. But these points are to be borne in mind: avoid all stimulation, and administer alteratives, such as arsenic, mercury, and iodide of potas-

sium, with a view to eliminating the possible specific origin, and then take that degree of rest and avoidance of violent exercise as favor the conservative powers of nature. Many of the symptoms of thoracic and abdominal aortic aneurism, and much of the suffering, are due to pressure by enlargement of the tumor. Sometimes the pressure becomes so great that the tumor ulcerates through into the spinal column, and again through the rib, at other times involving vital organs. Pressure upon the recurrent laryngeal nerve often produces a violent metallic cough, and that is a feature which should be borne in mind. With care, proper food, and avoiding muscular effort the patient we have here may be good for very many years. He is comparatively a young man, but the trouble has been that he has lived too rapidly, and, of course, if he has lived for fifty-four years and lived rapidly, the world does not owe him anything.

## AORTIC DISEASE OF THE HEART.

CLINICAL LECTURE DELIVERED AT THE MANCHESTER ROYAL INFIRMARY.

BY GRAHAM STEELL, M.D. (Edin.), F.R.C.P. (Lond.),

Physician to the Manchester Royal Infirmary, and Lecturer on Diseases of the Heart  
and in Clinical Medicine, Owens College, Manchester, England.

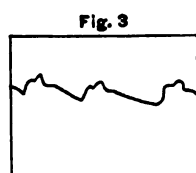
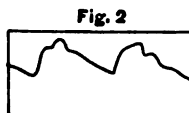
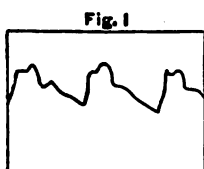
---

GENTLEMEN,—Aortic stenosis is most commonly a consequence of rheumatic endocarditis, and consequently we shall usually obtain a history of rheumatic fever in the course of the patient's life. Atheromatous aortitis is, in some cases, however, responsible for the lesion,—the morbid process involving the valves. In a very large proportion of cases of aortic stenosis there is some leakage through the narrowed orifice during diastole, but such regurgitation is seldom great when the stenosis is pronounced. In considering the physical signs presented by cases of aortic stenosis, I will begin with the auscultatory, although, as a general rule, inspection, palpation, and percussion should precede auscultation. The special auscultatory sign produced by the lesion is one in which, standing alone, very little confidence can be placed. The sign I allude to is, of course, a systolic murmur, having its maximum intensity in the aortic area. Technically, by the aortic area we mean the sternal end of the second right costal cartilage; but we must bear in mind the course of the aorta, as it dips into the very centre of the heart, to form, as it were, the great stem of the organ. The sternal end of the second right costal cartilage is named the "aortic area" because we can here best *isolate* aortic sounds or murmurs. In the case of an obstructive murmur, such a murmur is well conducted onwards in the course of the circulation from its seat of production, and so is loud in this area. The murmur is by itself of little diagnostic value for the following reasons: Even when obstruction may correctly be said to exist, such obstruction may be nominal only, and of absolutely no importance in the way of offering any impediment to the circulation; a little thickening of a valve or a tiny nodule projecting from its

surface may develop a loud murmur. Again, in cases of simple anæmia such a murmur is common enough, usually accompanied by a similar one over the pulmonary artery. Lastly, the auscultatory sign in question may be developed in great intensity when the aortic orifice, far from being less than normal, is actually increased in size. This happens when the aorta is dilated and its orifice correspondingly altered, though to a less extent than the channel beyond. The result is that the blood-current after passing through the dilated orifice spreads out into the still wider channel of the vessel; the condition of all others we know to be the great source of murmur production. If, then, a systolic murmur loud in the aortic area may be met with alike when the aortic orifice is normal in size, and when it is increased in size as well as when it is diminished in size, such murmur by itself can hardly be held to have diagnostic value as a sign of stenosis. I have said "by itself," however. No doubt the extreme loudness and harshness of such a murmur must receive attention; and when these properties are pronounced, aid in diagnosis may often be obtained from another physical method,—namely, palpation; for a marked thrill, corresponding to the murmur, is felt in the aortic area, and more or less over the course of the vessel. But thrill may accompany the opposite condition,—dilatation of the aorta, inclusive of its orifice,—such dilatation bringing the vessel in contact with the chest-wall to a larger extent than is normal. In such cases, visible pulsation will usually render the dilatation of the aorta evident, and the fact of dilatation will in some degree oppose the idea of there being material stenosis of the orifice. The two conditions may, however, actually coexist, as I have seen. I have spoken of the loudness and harshness that often characterize the murmur of aortic stenosis; but, further, the murmur often strikes one as being of remarkable duration, while the pulse is characteristically infrequent and sluggish. One is reminded of the breath-sounds in glottic obstruction, each of which is of such duration as to forbid frequency of respiration.

An important diagnostic feature of the murmur of aortic obstruction is the absence, or at least deficiency, of the aortic second sound that should follow the systolic murmur. Such absence or deficiency of the physiological second sound is readily explicable when we consider the lesion and its effects. Lastly, a diastolic murmur, seldom loud, but not rarely, quite distinct, high-pitched, and "whiffling," is often audible, indicating a measure of incompetence.

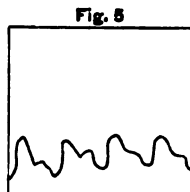
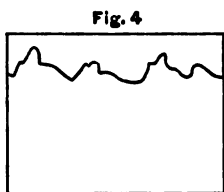
There are two varieties of pulse of diagnostic value in relation with aortic stenosis. Neither is pathognomonic; yet in combination with a loud, harsh, systolic murmur, accompanied by a thrill in the aortic region, the second sound in the same region being deficient or absent, either of the pulses to be described renders the diagnosis of aortic stenosis almost secure. The two pulses are usually termed the anacrotic pulse and the bisferiens pulse. The former is, perhaps,



FIGS. 1, 2, and 3.—Typical tracings in aortic stenosis.

of the greater diagnostic value. It gives a sphygmogram with the following characters,—the upstroke sloping, the summit of the curve formed not by the percussion wave, but by the tidal wave, and the dicrotic wave usually ill-developed. (Figs. 1, 2, and 3.)

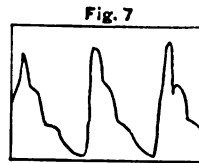
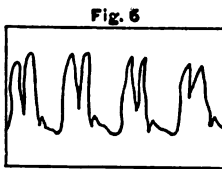
The percussion wave can hardly be said to exist, but its situation is indicated by a slight notch at the beginning of the tidal wave. You may imagine for yourselves what will be the sensation conveyed



FIGS. 4 and 5.—Showing two types of pulse observed repeatedly at different times in the same case. In Fig. 4 the tracing was taken while the patient was under the influence of digitalis, and a tendency to assumption of "bigeminal" character is shown in the pulse.

on placing your finger on such a pulse, and exerting pressure upon it. Those who disparage the serviceableness of the sphygmograph can hardly deny its value in "educating the finger," as the phrase is. Even after long experience in appreciating pulse conditions, it is at times necessary to check the impressions received through the finger by a permanent record of the pulse condition at times at which it was taken. The other type of pulse associated with aortic stenosis

is, as I have said, the bisferiens pulse (Fig. 6), indicating its most marked feature, "twice striking;" for, when one places one's finger on an example of this pulse, one is aware of a double beat. I do not think, however, with the finger alone, one could decide which of the waves was so developed as to give the impression of the second stroke. By far the most common "second beat" of the pulse perceptible by the use of the finger only is the dicrotic wave in febrile conditions. In aortic disease, both stenosis and incompetence, the dicrotic wave is apt to be deficient; so that it is very unlikely that the dicrotic wave should be so developed as to give rise to a "second beat." If, then, we feel a double pulse-beat in a case we know to be one of aortic disease, the presumption should be that the pulse is not a so-called dicrotic one, but is "bisferiens." (The bisferiens pulse, of course, can hardly be mistaken for a bigeminal one (Fig. 5), in which each beat represents a separate systole.) In the bis-



FIGS. 6 and 7.—Both from the same patient, right and left radials respectively, showing difference referred to below.

feriens pulse, the second beat represents the tidal wave which in the sphygmogram is seen to start low down, near the "respiratory line," and to be sharp and angular in place of being rounded and sustained.

A curious feature of this pulse is the development of its peculiar characters on one side only. In fatal cases of the kind, we have entirely failed to discover the cause for such a difference between the radial pulses. (Figs. 6 and 7.) Now, all degrees of stenosis are met with, and the incompetence that is so frequently associated with stenosis is also met with in all degrees. The influence of such incompetence upon the typical sphygmogram of pure stenosis will of course vary. I recently had a case of the combined lesion under my care, in which, though the murmurs of stenosis and incompetence were both loud,—the murmur of the former being accompanied by a thrill,—the sphygmogram was typical neither of stenosis nor incompetence; the one lesion had neutralized the other. (Fig. 9.)

With regard to pulses supposed to be characteristic of lesions,



it is well to bear in mind that the pulse is a vital phenomenon, and is under the influence of a variety of circumstances, so that it is subject to great variations in the course of the same case. I show you, in illustration, tracings taken from the same patient when her pulse was frequent and when it was the pulse typical of aortic stenosis.

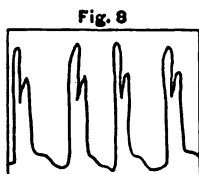


FIG. 8.—A less developed bifurcated pulse. A marked thrill and loud systolic murmur were present in the aortic area.

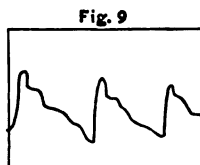


FIG. 9.—Tracing referred to in the text as the case in which the two lesions—obstruction and incompetence—seemed to neutralize one another.

The same might also be said of the murmur that indicated the lesion, for when the pulse was frequent the murmur presented no special loudness. In the sphygmogram of the frequent pulse, you will notice, however, one feature that might suggest aortic stenosis,—the sloping upstroke.

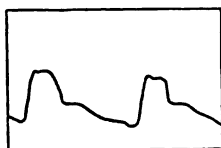


FIG. 10.—Tracing from case of dilated aorta with slight incompetence of valves.

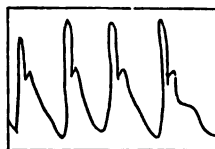


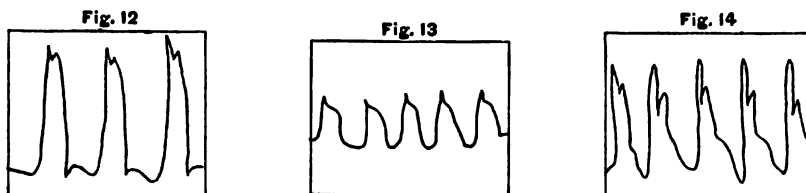
FIG. 11.—Typical tracing of aortic incompetence. Case referred to in text. Although this case was evidently of vascular (aortic) origin and syphilitic, the history of rheumatism five years before the patient's death led to the lesion during life being regarded as rheumatic in origin. This tracing shows that the sphygmograph cannot distinguish endocarditic cases from atheromatous or vascular cases, as has been supposed.

There are certain physical considerations that have a very important bearing on our appreciation of the results of valve-lesions generally, and of aortic stenosis specially. First of all, it is evident that in the immense majority of cases aortic stenosis is a slowly produced lesion. The acute production of obstruction by a thrombus is an excessively rare event, and when it occurs, the element of ob-

struction plays but a minor part; the conditions that cause the formation of a thrombus so overshadow its mechanical effects.

Granting that the ordinary lesion is slowly produced, the left ventricle has plenty of time to accommodate itself to the abnormal amount of work thrown upon it; moreover, it meets the difficulty, with which it has to contend, during systole only, and hypertrophy is all that is necessary for compensation. There is no occasion for any dilatation of the chamber so long as the left ventricle remains healthy and vigorous,—i.e., able to complete its systole. Let us suppose, however, that from one cause or another the nutrition of the heart-muscle fails; that some degree of muscle-failure sets in. Such failure eventuates in one or other, or in both of two abnormal conditions, the ventricle ceases to complete its systole, or the mitral valves cease to perform their function in a perfect manner, so that regurgitation into the auricle is permitted. These two results of muscle-failure may, and commonly do, occur together. Now, the moment one or other is established, there is at the same time established a tendency to increase of the capacity of the left ventricle,—that unmixed evil in cardiac pathology, dilatation. Let us inquire farther into the matter. It is a physiological fact that, if the ventricle is made to contend in the performance of its function against an obstacle too great for its strength, the result is an increase of the residual blood that in health occupies only the suprapapillary space immediately below the valve-curtains. It is not true that in health the ventricle wrings out every drop of blood; but physiologically the residual blood is limited to the small suprapapillary space, where probably it has a purpose concerned with the function of the valve-curtains. But this abnormal state of imperfect systole—systole-catalectic I have called it—is not all. It seems to be a physiological law that if the ventricle is strained beyond its strength, systole-catalectic is not the whole of the evil that results, the mischief extending to the diastole of the ventricle, which becomes excessive. Dilatation, I have called an unmixed evil in cardiac pathology, and I fancy no one will be inclined to dispute its title to the term. We have traced the consequences of imperfect systole, the result of muscle-failure, to this unmixed evil of dilatation; let us do the same in the case of the other result of muscle-failure, namely, mitral incompetence. The mitral valves are dependent on muscle contraction for their perfect function; muscle contraction is a necessary com-

plement of the valve apparatus. It is necessary for the perfect closure of the valves in two ways,—the orifice the valves have to close is diminished by such contraction, and the valve-curtains are supported in action by shortening of the papillary muscles.



FIGS. 12, 13, and 14.—Typical tracings of the pulse in aortic incompetence.

Having considered the mode of production of mitral incompetence, let us inquire next as to its effects. It will result that a certain amount of blood is returned to the auricle with each systole of the ventricle; so that the auricle has to receive during its diastole not only the normal amount of blood from the pulmonary circulation, but, in addition, the regurgitated blood from the ventricle. Both the normal and the abnormal amounts of blood must find their way to the ventricle during its succeeding diastole, if the circulation is to be maintained; and this implies increased capacity of the chamber, or dilatation.

We can easily understand how it happens that aortic stenosis is a lesion often borne without suffering during a long series of years, because, so long as the nutrition of the left ventricle is maintained, there is nothing to occasion dilatation. It is only when the nutrition of the left ventricle fails from one cause or another, and systole-catalectic becomes established or mitral incompetence results, that dilatation is produced. Impairment of the heart-muscle may result from general causes, as anæmia and pyrexia, or from local ones, such as disease of the coronary arteries and adherent pericardium; but, whatever its cause, its influence in the case of a heart handicapped by a lesion like aortic stenosis must be disastrous. All treatment, hygienic and medicinal, should be carried out in the light of these considerations. The lesion, once established, probably cannot be altered by treatment; but an immense amount may be done by treatment in the way of aiding the heart to bear with impunity its extra burden, and so to maintain, practically unimpaired, the general circulation of the blood.

In our consideration of the effects of aortic stenosis on the left ventricle of the heart, we have found evidence that hypertrophy is the only change necessary for compensation of the lesion, and that dilatation only results when muscle-failure of the heart has set in, interfering with the completion of the systole of the left ventricle, or with the function of the mitral valves. In the case of aortic incompetence, the effects of the lesion upon the ventricle must be very different in the first instance. They must be exerted at first during diastole only. Blood is pouring into the ventricle from the aorta as well as from the auricle, and increased accommodation must be the first requirement, implying dilatation of the left ventricle. But the larger a chamber of the heart becomes, the greater is the demand upon its muscle-walls; therefore urgent need of hypertrophy arises.

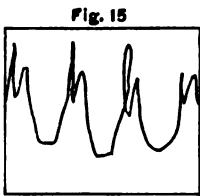


FIG. 15.—Shows how a loop is sometimes formed by the tracing of the percussion wave in cases of free regurgitation, so great is the jerk of the lever.

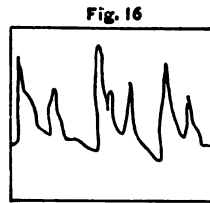


FIG. 16.—Bigeminal pulse from case of aortic incompetence under the influence of digitalis.

The contrast between the effects of aortic stenosis and aortic incompetence upon the left ventricle is, as you will see, a very marked one. In the case of stenosis, it is obvious that hypertrophy is all that is necessary to establish compensation; and dilatation is a result of muscle-failure only, while, in the case of incompetence, dilatation is the first change, and the direct tendency to it is always present, although held in check by hypertrophy of the ventricular wall.

In comparison with aortic incompetence, we can well understand from these considerations the good prognosis that has long been attached to aortic stenosis; but apart from the fact that the two lesions are often combined, it must be admitted that even pure stenosis is a grave lesion, and that it is only borne with comparative impunity so long as the nutrition of the heart-muscle is well maintained. When this has failed from any cause, the prognosis is bad, and sudden death, in my experience, is not rare.

The signs of aortic incompetence are many and characteristic,

and I must limit my observations to a few comments upon them. First of all, the visibleness of arterial pulsation is a striking feature. It is most characteristically seen in the carotids, high up in the neck, at the angle of the jaw; but in extreme cases all the arteries in the body show it; even comparatively small peripheral vessels becoming visible in this way. The root of the neck shows best any venous pulsation that may be present. Its "double" character usually enables it to be at once distinguished from the single arterial throb; but if there is doubt, light pressure at the base of the neck at once obliterates the venous pulse, which may be exceptionally single and systolic in rhythm, owing to paralytic distention of the right auricle. In aortic incompetence, grasping the fore arm enables us to appreciate the exaggeration of arterial pulsation. A linear erythema made by friction on the skin will, again, show capillary pulsation. The sphygmogram is characterized by exaggeration of the percussion wave and deficiency of the dicrotic one. The tidal wave is usually not sustained; that is to say, no sooner is it formed than it collapses, and it often assumes angular shape. There are several different types met with, depending on the degree of incompetence, and the circumstances in which it arises. Thus, a slight leakage because of dilatation of the aorta may show in the sphygmogram a well-sustained tidal wave, little exaggeration of the percussion wave, and fair development of the dicrotic wave; little or nothing, in fact, that is characteristic. I have already referred to the influence on the pulse exerted by coexisting aortic stenosis; so that the features characteristic of each lesion may be blended in all proportions.

With regard to auscultation, the special murmur of the lesion is, of course, a diastolic one, conducted downwards and towards the left, over the ventricles, and often reaching the apex. The murmur may be of extraordinary loudness, and even audible at a short distance from the patient; it is then generally musical. On the other hand, the murmur may be feeble and curiously limited, apparently capriciously so. Last of all, it may temporarily disappear under certain circumstances,—especially acute febrile disease and great disturbance of the circulation,—although the jerking arterial pulsation remains as before, and no doubt the incompetence also. It has happened to me on more than one occasion to be able to hear the diastolic murmur in the femoral artery, to be described immediately, when I was unable to identify any diastolic murmur over the heart

itself, although later such a murmur became plain enough. Before we are entitled to say that no diastolic murmur is present over the heart, it is necessary to explore the whole of a triangular region defined as follows,—by a line drawn along the right border of the sternum from the second right costal cartilage to the xiphoid, and by lines drawn from the apex-beat to the extremities of this line. The diastolic murmur of aortic incompetence may be loud over all this area, or, on the other hand, it may be limited to any part of it.

In nearly all cases of aortic incompetence, the diastolic murmur is accompanied by a systolic one in the aortic region at least. This by no means necessarily, as we have seen, indicates stenosis. We have already considered the dilatation of the left ventricle that results from the lesion; and if the left ventricle retains its vigor, the aorta will suffer distention during the systole of the ventricle. This will tend to the production of dilatation of the vessel, an all-sufficient cause of systolic murmur.

Again, mitral and tricuspid incompetence without valve-lesion is a common result of muscle-failure in cases of aortic incompetence; or, aortic incompetence may be associated with mitral valve-lesion produced by the same rheumatic endocarditis that crippled the aortic valves. In subjects over middle age, with a history of rheumatic fever in earlier life, I have more than once met with aortic incompetence entirely independent of rheumatic endocarditis and belonging to the common “degenerative” (atheromatous) type; the history proving deceptive, so far as pathological diagnosis was concerned.

In the case from which Fig. 11 was taken, syphilis had produced disease of the aortic coats, quite independently of a rheumatic endocarditis that had occurred five years before, the effects of which latter were barely visible on the mitral valves.

Attention to the physiological heart-sounds, when present, should always precede the notice of murmurs. Accentuation of the aortic sound often throws great light upon the origin and nature of a given case of aortic incompetence pointing to disease of the aorta, rather than of its valves. On the other hand, entire absence of the second sound suggests great damage to the valve-structures, and, in an acute case, may become a point in the diagnosis of septic endocarditis.

I have already referred to a diastolic murmur in the femoral artery. In a pronounced case of aortic incompetence, when one

listens over the vessel, avoiding pressure with the stethoscope, one hears a systolic (*i.e.*, with reference to the heart) sound, and more closely resembling a cardiac "sound" than the dull "thud" normally heard over the vessel. The sudden distention of the arterial coats that must take place in aortic incompetence readily explains the difference. The moment we exert pressure on the vessel, however, a murmur, as in health, is produced, either accompanying or replacing the sound. Sometimes the sound disappears at once with the production of the murmur; at other times it is difficult to annul the sound by ordinary pressure. Further, at a certain degree of pressure a diastolic murmur—and it is this that is characteristic of free aortic regurgitation—becomes audible. In certain cases this diastolic murmur is developed by very slight pressure; in others it is only audible at a particular degree of pressure which may be readily passed, so that the murmur is missed in the passage from light to heavy pressure.

Lastly, in very rare cases of great incompetence two sounds, not murmurs, become audible over the femoral artery, pressure with the stethoscope being avoided. The phenomenon was first described by Traube, of Berlin, many years ago. There can be no question of its occurrence, although its explanation is not evident. I have recorded a good instance of this rare physical sign in the proceedings of the Manchester Clinical Society.

Time will not permit the discussion in detail of the various forms of disease that eventuate in incompetence of the aortic valves, though some have been incidentally mentioned. Again, the important bearing of implication of the coronary orifices in disease of the aorta (often giving rise to angina-pectoris) on the nutrition of the heart must not be lost sight of.

## **PULMONARY TUBERCULOSIS; SPECIFIC PHARYNGITIS; AORTIC REGURGITATION.**

CLINICAL LECTURE DELIVERED AT THE RUSH MEDICAL COLLEGE.

**BY E. FLETCHER INGALS, M.D.,**

Professor of Laryngology in Rush Medical College; Professor of Diseases of the Throat and Chest, Woman's Medical College; Physician and Surgeon to the Central Free Dispensary for Diseases of the Chest, Throat, and Nasal Cavities, Chicago, Illinois.

---

GENTLEMEN,—This man is thirty years of age, and has been sick since the middle of last February. He was perfectly well before that. His symptoms, he says, at the beginning, were fever and chills every day, which occurred at no regular time, but generally in the morning about seven o'clock, as soon as he got out of bed; he had a hard chill and the fever followed it, lasting several hours. He had pain in his chest all the time, mostly on the left side. He had no pain in the throat, but the roof of the mouth got very dry. His trouble ameliorated, but he has been feeling badly ever since his first attack. Recently he has been hoarse; says he took a severe cold about a week ago; has no appetite; has lost flesh; formerly weighed one hundred and thirty-eight, now weighs one hundred and eighteen pounds; has pain in the stomach; it seems to him as if the trouble was in the liver or kidneys or both; he has difficulty at times in passing his urine, which is of high color. The commencement of his illness seems to have been an attack of influenza. There is no history of throat trouble at that time, so I judge that any trouble in that part, if present, has been but recent. The history he gives is quite characteristic of a large number of patients I have seen during the last year. They have been troubled with influenza and slowly decline in health, and afterwards show signs of pulmonary disease. I have not examined this man's chest, but the loss of flesh, the fever, the expectoration,—between a pint and a quart, he says,—all these point strongly to the pulmonary disease. I find his pulse 142. I am coming more



and more to attach importance to a rapid pulse in a person who has been sick for any length of time. It is very seldom that a person comes to me who has been sick a long time with a pulse at 120 that I do not find a cause for it in the lungs. Of course there may be other causes, but the class of patients who come to me complain of trouble with the lungs rather than anywhere else. His skin is normal now; his tongue is natural; there is nothing there to indicate the nature of his disease. In order to be sure of our diagnosis, of course the chest must be examined. I think you can, at a distance, notice the difference in the resonance of the two sides of the chest; above the second rib the resonance is dull and of high pitch as compared with that below, and as compared with the resonance on the left side. In examining a chest where you suspect that there is tubercular trouble, you must note first the respiratory movements. Sometimes you will get a good deal of information from that. You will find that one side moves less than the other. In this chest there is not so much difference as you will often note, but the dulness you get from percussion indicates that there is something wrong. This dulness is over the right apex. Upon examining the chest with the stethoscope, always listen at some part where you think it will be normal. When I carry my examination upward towards the apex, I find fine, moist, crackling sounds; carrying it a little higher to the lower margin of the second rib, and above this, I find that the sounds are harsh and there are some moist sounds, not so fine as those heard below. The voice sounds are more distinct than below. All these things indicate consolidation. In this case I do not think you will be able to detect dulness on the left side. I should not feel safe in saying that there is dulness as compared with the right side, but there are no two chests alike. We have found out, then, that there is consolidation at the apex of this right lung, and, taking this in connection with the symptoms, there can be no question whatever as to the nature of this man's disease. Whether it has affected the throat or not we will see. In this case there is nothing that is characteristic about the throat, but as we look into the patient's mouth we observe a peculiar pallor of the uvula extending away forward to the hard palate. The vocal cords and larynx are healthy in appearance. This is the condition in some cases of tuberculosis where the larynx is somewhat involved. There is no evidence whatever of tuberculosis in the larynx, except the pallor. In some of these

cases there is partial paralysis as the result of the action of the nerve supplying the larynx. It happens often that you will find pallor and a little hoarseness, as in this case, even if there is no decided evidence of disease. In some of the cases they will go from this state on to a decidedly serious condition within from four to eight weeks. In some others you will find simple congestion of one or more of the parts, while in others there will be a uniform congestion all over. But none of these can you distinguish from catarrhal congestion or a simple laryngitis. In this case there can be no doubt about the true nature of the disease. The question of treatment simply resolves itself into treatment for pulmonary trouble. Local treatment is not of so great importance as is sometimes supposed. If the general condition can be improved, you will be likely to improve the larynx. In a case showing no more than this I see no reason to make local applications at all. Internally I should use such remedies as would be likely to relieve the pulmonary trouble, and I have very great confidence, myself, in two or three remedies. I believe that the chloride of calcium is one of the best remedies we have for checking pulmonary tuberculosis. I have very often seen it checked, and I feel confident that it was due to this remedy. I should recommend here alcoholic stimulation also. But aside from these you will nearly always have to look to the digestion; bitter tonics and digestive adjuvants; and something to keep down the fever. If we give the patient opiates to check the cough we are likely to render the digestive functions almost inert; and therefore I avoid opiates whenever it is possible, and rely more upon the remedies like hyoscyamus, camphor, cannabis indica, etc., with one-third of a grain of nux vomica as a tonic, sometimes using quinine to check fever. We will recommend him to use alcoholics with his meals,—as much as he can take without feeling it in the head,—and between meals, in milk, but never alone. The chloride of calcium may be taken in whiskey,—ten grains in a drachm of whiskey.

#### SPECIFIC PHARYNGITIS.

This woman has been in bad health for about two months; does not feel like working; does not feel really sick, but is not well. She has some appetite, but the act of swallowing gives her very great pain. The pain is in the throat at the upper portion of the pharynx. There is a little enlargement of the glands just behind the angle of

the jaw; none elsewhere. She is not troubled with cough, has but slight fever, has not lost flesh, and has lost but little strength, if any. She has had an eruption upon the limbs for a couple of months, which fails to heal. She has soreness of the right nostril, with a dark scab upon it. The pulse is about 100. We can get nothing more out of the history. She is a widow, has had eight children, the last one four years ago. I am unable to see her throat. She retches so that I cannot see the whole of the palate, and do not see anything that would indicate the true nature of the disease. She says there is no reason for the soreness of the nostril except her bad blood. I find it extends up into the nostril three-eighths of an inch on the outer part, the septum not being at all involved. Upon the right leg we find four different sores, scaly upon the surface, which have never broken out. While there is nothing characteristic about these and nothing absolutely characteristic about the appearance of the nose, I do not think there can be any difficulty in making a correct diagnosis; and we will put it down as specific in character. Iodides will be better than anything else. Ten grains of potassium iodide, increased to twenty or thirty grains at a dose, three times a day. In giving the remedy it is easier taken in water largely diluted, and we order it taken with a full glass of water at each dose. This medicine she must continue for a number of months.

#### AORTIC REGURGITATION.

Here is a man who for years has had a peculiar sound of his heart, which he can hear himself. The heart is quite regular most of the time, but now and then intermits, perhaps once in a couple of minutes. I saw him a few days ago, but did not go carefully into the history of the case. He says he is somewhat short of breath after any exertion, and he has been so troubled for some years. He does not know that he has ever had rheumatism.

*Question:* Did you ever have any serious illness of any sort?

*Answer:* I was laid up in bed about twelve years ago. The trouble, whatever it was, came on of itself,—might have been from exposure; I believe the doctors concluded it was paralysis of the spine.

*Question:* Are you troubled with cough?

*Answer:* I was troubled with cough last summer, but this winter I have not had any cough at all, until two weeks ago I had the grip.

He has no cough; he has no symptoms to indicate trouble with the heart; he is a little short of breath, but to a very trifling extent; he has no pain. The heart is evidently of normal size, in the natural position, beats about seventy-five per minute, and regularly, excepting it drops a beat once perhaps in two minutes. In listening at the apex I find no abnormal sounds, excepting a peculiar cooing sound, which I heard sometimes singly, sometimes two or three times together, and I seemed to hear it with the diastole of the heart. When I heard it first I thought it was the systole, but to-day I am satisfied it is not systolic. By placing my finger upon the carotid pulse I get two or three ordinary beats, then I hear that cooing sound, then two or three more beats, then four or five cooing sounds, then as at first again. When this cooing sound is present it is so loud as to mask everything else, but when it is absent I hear a murmuring sound, which is loudest in the aortic area. The cooing sound seems, therefore, connected with some lesion of the aortic valve. The cooing sound is not present more than two-fifths of the time, and when it is absent I can hear a to-and-fro murmur, showing that there is both murmur and regurgitation at the aortic valve. I do not find it quite so loud to-day as the last time I listened to it, but so loud that it may be readily heard. I would have you notice that it can be heard all over the chest. All heart-murmurs are heard loudest in certain areas. This one is heard loudest in the aortic area. The amount of regurgitation is not very great. We can recognize it slightly by the pulse. When his arm is hanging we get a full stroke, but by raising the arm we obtain a more distinct stroke, termed the hammer-pulse, though not very intense. It is a case of aortic regurgitation, but as long as there is no hypertrophy or dilatation of the heart the patient's general health is not affected. Generally there is enough obstruction, so that the heart hypertrophies and dilates, and eventually dilatation becomes too great and compensation cannot be accomplished. At present compensation is nearly perfect. He has about seventy to eighty pulsations per minute, very little difficulty in breathing, and no treatment is necessary at present.

## TUBERCULAR PLEURISY; TACHYCARDIA.

CLINICAL LECTURE DELIVERED AT THE CITY AND COUNTY HOSPITAL, SAN FRANCISCO.

BY J. O. HIRSCHFELDER, M.D.

---

GENTLEMEN,—The patient who will be the subject of our clinic this morning is John Nagle, aged thirty-two, a native of Ireland, and a laborer. He was admitted to the hospital May 8, 1891. His father died of unknown causes. Two of his sisters were killed by an accident. His mother, two brothers, and six sisters are living and well. There is no history of any hereditary disease in the family. The patient has had the usual diseases of childhood. In November, 1883, he had gonorrhœa, which lasted for six weeks. In October, 1884, while at work, he was struck over the inner angle of the left orbit with a piece of steel, which produced a lacerated wound of the scalp and comminuted fracture of the skull, several small fragments of bone being removed; the wound was stitched, and the patient became convalescent in two months. The present trouble began during the latter part of March, 1891, with loss of appetite, which continued, accompanied by loss of flesh, until the latter part of April, when the patient began to cough, becoming weaker and weaker, the loss of appetite being more marked, and emaciation to the extent of twenty pounds' loss of weight in two months setting in, accompanied by occasional night-sweats and an expectoration, small in quantity and muco-purulent in character. One week ago the patient had hæmoptysis, which lasted two or three days. He has been in the habit of using tobacco and alcohol moderately. At no time has he complained of shortness of breath.

As far as we can learn this patient in March began to be affected with cough, loss of appetite, and expectoration of a small quantity of muco-purulent matter, followed then by hæmoptysis. During all this time there has been no marked shortness of breath; no

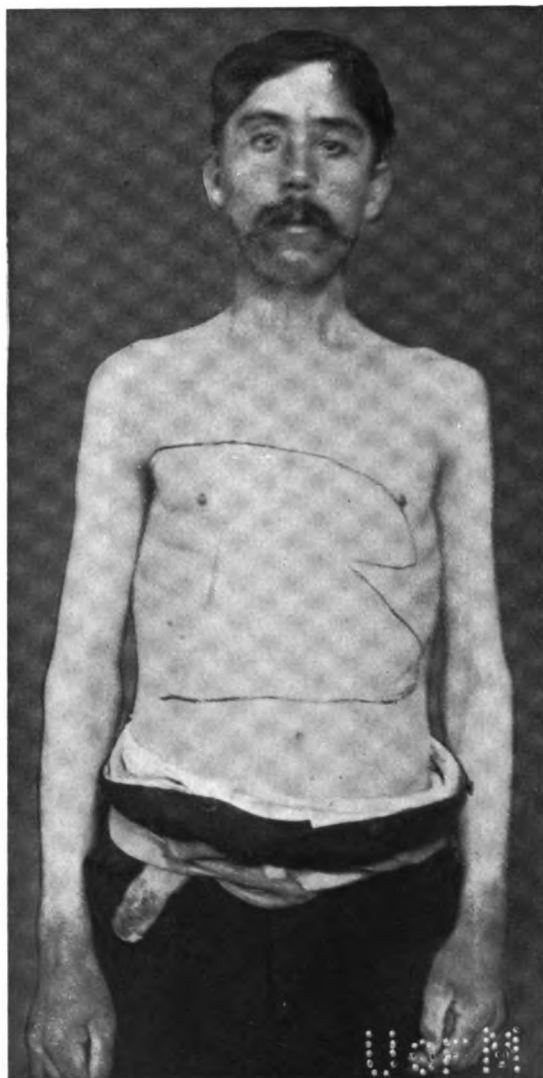
dyspnœa. The patient looks somewhat pale, but with the exception of that pallor, there is really nothing which is striking in his appearance.

The history which you have just heard read directs your attention to the lungs. Loss of weight, night-sweats, expectoration of muco-purulent matter, followed by hæmoptysis, is a history that would direct your attention certainly to the lungs. You have heard, however, that there are no diseases of the lungs in this man's family. No member of his family, so far as we have learned, has had consumption; no one has died of that disease. The chest is long, moderately broad, moderately deep, and moderately well arched. So, then, with the exception of being somewhat longer than would correspond to the height of the individual, the chest in form is practically a normal one. You observe no marked depressions of the supraclavicular and infraclavicular fossæ, but they are rather longer upon the right side than upon the left. There seems to be no marked difference otherwise between the two halves of the chest wall. The character of his breathing is costo-abdominal, with a preponderance of the abdominal factor in the respiration. Upon the patient taking a deep breath, you will, if you will observe the mark that I have made upon the chest, notice that upon the left side it moves a great deal more, upon forced respiration, than it does upon the right. And the same difference that was observed above where the mark has been made, is more evident below. The entire right side of the chest moves less than the left.

Upon the right side, anteriorly, in the supraclavicular fossa, the tone, as compared to the left side, is slightly dull. Upon the clavicle the difference is likewise present, and the same. In the second intercostal space upon the right side the tone is tympanic and dull. Here in the mammary line, at the lower border of the fourth rib, the tone becomes flat. Posteriorly, there is dulness of the left apex as low as the lower border of the first dorsal vertebra; upon the right side the dulness extends down as far as the upper border of the first dorsal vertebra. Then there is a clear space upon the right side, the clear space extending down to the seventh dorsal vertebra, and from the seventh dorsal vertebra down posteriorly the tone is flat. You see that the height anteriorly is about the same as the height posteriorly. Now, upon the left side of the thorax you find that the cardiac dulness is continuous with the dulness that we had found

upon the right side; that the cardiac dulness extended over in the sixth intercostal space three centimetres to the left of the mammary line. You see that the liver dulness extends downward to three and one half centimetres above the umbilicus, and extends to the left fourteen centimetres from the median line, and you see that the area of cardiac dulness is separated from the area of liver dulness by a clear space. (Fig. 1.) The spleen, you observe, does not quite reach to the anterior axillary line. Hence you know that the spleen is not enlarged. Anteriorly at the right apex you hear bronchial inspiration and bronchial expiration, together with numerous dry râles. Posteriorly you hear the same, but more marked. At the left apex you hear, likewise, bronchial inspiration and bronchial expiration. On the right side, over the region of dulness, you hear hardly any respiratory murmur; upon the opposite side you hear the normal vesicular respiration. You notice that when the patient bends over horizontally the dulness is not as apparent where it was present before, although it does not become perfectly clear. Here, anteriorly, above the region where the liver is probably situated, we had absolute dulness extending up to the lower border of the fourth rib, and when the patient is placed in a reclining position the tone becomes clear. The dulness upon the right side changes with any change of position. Let us see whether the dulness that corresponds to the area of cardiac dulness does the same. In point of fact, you will notice that that is the case. In a sitting position we have absolute dulness. When the patient lies down, the tone is clear, and the area of cardiac dulness, instead of extending three centimetres to the left of the mammary line, is now found at the mammary line itself. The expectoration, as you see, is a greenish-yellow, mucopurulent sputum, and in addition to that there is one sputum that is tinged slightly brown with blood. A specimen of this sputum has been prepared, stained according to Ehrlich's method with methylene blue and fuchsine, and it is now under the microscope for your inspection.

In the case of our patient at a previous clinic we found dulness of the apices on both sides, with bronchial respiration and a few bronchial râles. Besides this dulness of both apices, we found a region of dulness that is still marked off at the lower portion of the right side of the chest which was continuous with the cardiac dulness, and this cardiac dulness extended over three centimetres to



**FIG. 1.—Areas of percussion dulness of the heart, liver, and pleuritic exudation.**





the left of the mammary line; that when the patient changed his position, the position of this dulness in the lower portion of the chest changed, and likewise the area of cardiac dulness changed when he lay down, not extending by any means as far to the left as it had done in the erect position. We examined the sputum under the microscope, and found that it contained a large number of the bacilli of tuberculosis. The question for us this morning is to translate the symptoms that we found here into their pathological equivalent; in other words, to make the diagnosis of the condition present. We have found dulness of both apices, with bronchial inspiration and expiration, with an expectoration of muco-purulent sputum containing the bacillus of tuberculosis. This means a tubercular infiltration of both apices. Whenever you find a dulness such as we have found here, which changes its position as the position of the patient changes, and with the degree that it has changed here, there must be fluid in the chest cavity; there is nothing else that can do that. When you listen to the respiratory sound over the region of dulness, compared with the respiratory sound upon the other side, you hear a very much diminished murmur upon the right side as compared to the left, showing that there is something between the lung, we will say, and the chest wall which prevents the transmission of the sound. When a fluid is between the lung and the chest wall the transmission of the sound produced is diminished, because the air does not enter into the lung with the same degree that it does upon the other side, and there is likewise something which prevents that sound which is produced from reaching the ear. Thus you have two reasons why the respiratory murmur is less upon the right side than upon the left. So, then, all signs point to fluid, but in forming an opinion it is necessary or valuable to know what the nature of the fluid is. It will make a great difference both in our treatment and in our prognosis whether this fluid is a clear limpid fluid, or whether it be pus. The symptom of Baccelli will aid us in forming an opinion as to the character of the fluid. Baccelli's symptom is that the clearer the fluid that forms the pleuritic exudation the clearer the whispered voice will be transmitted through it to the ear. A number of investigators have disputed the correctness of Baccelli's opinion. My observation thus far has been confirmatory of Baccelli's statement, and the reason why a whispered voice should be clearer in a clear fluid than in a turbid

fluid is a very simple one. In order that a tone should be a clear tone it should be composed of simple factors. A noise is a combination of discordant notes; a combination of sounds whose vibrations are not in harmony one with the other. The more a tone is separated from other tones, the clearer it is. If one person speaks what he says, the sounds that he makes are clear and distinct. If half a dozen people are speaking at the same time, the result is a noise, a confusion, an obscurity of sound. Now, when the whispered voice passes through a fluid, in passing through a clear fluid all the vibrations pass at the same rate through all parts of the liquid. But, if we have a turbid fluid, that is to say, a fluid that is composed partly of liquid and partly of solid, vibrations would not pass through the liquid and through the solid at the same time. Vibrations passing through the solid would reach the chest wall sooner than vibrations which pass through the liquid. Consequently, at the chest wall we would get delayed vibrations and vibrations not delayed. In other words, we would get different sounds at one and the same time.

Suppose the sounds produced by A, B, C, and D, pronounced one after the other rapidly, for the sake of illustration; and these sounds A, B, C, D were transmuted through a turbid fluid; and supposing that the sounds were such that, by the time the sound A reached the chest wall in passing through the liquid the sound B would reach the chest wall in passing through the solid constituents, and by the time that the sound B would reach the chest wall passing through the liquid the sound C would reach it passing through the solids, and that by the time the sound C had reached the chest wall passing through the liquid the sound D had reached it passing through the solid. You see the result of such an arrangement would be that all of these various sounds would be heard together in confusion. Supposing there were only two sounds, the sounds A and B; and supposing that by the time the sound A in passing through the liquid reached the chest wall the sound B, which we suppose is produced later, reached the chest wall through the corpuscular elements; then, by the time the sound reached the chest wall, you would hear, instead of A and B one after another, the two sounds together, and instead of being clear sounds there would be a confusion of sound.

Then the clearer the fluid, the clearer the sound. Why a whispered sound and not a loud tone is required is because the loud

tone would reach you through the vibrations through the wall itself and through the air, and what you want to get is the sound which reaches you through the chest wall and not that which reaches you through the air. It is very hard to exclude the sound that comes through the air when the tones are loud. It is not the loudness of the tone, but the distinctness that we depend upon. In this case you hear clear and distinct, but not loud, sounds.

We have found, then, according to Baccelli's symptom, a clear fluid. Now, there is no objection to inserting the hypodermic needle to verify the correctness of our prognosis. And, in point of fact, you see the fluid is as clear as the tone that was heard: perfectly limpid. The diagnosis that we have made has been verified by our examination. We found tubercular infiltration of the apices with tubercular sputum. We found a clear fluid exudation, but there was one peculiar thing that demands explanation here. We heard a tympanitic tone above this pleuritic exudation. A tympanitic tone is a clear tone, a simple tone, a musical sound, where as a non-tympanitic tone is a noise. When you percuss over the normal lung you have different tones produced by the percussion, and these tones that are produced do not harmonize one with the other, and the result is that you have a discord, a non-tympanitic note. You will have a tympanitic note when the sound produced is caused by but one of these vibrating bodies. If you should percuss a lung taken out of the body, you would hear a non-tympanitic note, as you would get the audible vibrations from the alveolar walls and likewise from the air contained therein. If the alveolar membranes produce audible vibrations, and these little air-spaces produce audible vibrations also, the result is a confusion; but if the membrane is relaxed, or is infiltrated, or has lost its elasticity so that it cannot produce audible vibrations, then the only thing that causes these vibrations are these little air-bubbles, and they are the ones that cause the tympanitic note. A tympanitic tone is produced, first, by a relaxation of the alveolar walls so that their vibration does not produce sound. Secondly, it can be produced by an infiltration of that membrane, so that, being thick, it no longer produces those same audible vibrations. Or, the tympanitic tone can be produced by the lung tissue losing its elasticity, in consequence of which it does not vibrate as it did before and produce audible vibrations. The reason why we have a tympanitic tone here is that the lung is compressed by the fluid,

the air is squeezed out, the alveolar walls are not so tense, and the consequence is that when I percuss, the alveolar walls do not produce audible vibrations, and a clear instead of a non-tympanitic tone results. The reason that this man has no shortness of breath is, in the first place, that the quantity of fluid that is present there is not so enormous as one would think from the height to which the fluid reaches. When I had introduced my hypodermic needle a considerable distance into the area of dulness, no fluid came out, showing that it was not reached; as I withdrew the needle, fluid came. This shows that the layer of fluid is a small one, and, consequently, the quantity of lung tissue that is placed out of function is small. We utilize only a comparatively small portion of our lung in respiration. Under ordinary circumstances we do not inhale anything like the quantity of air that could enter our lung. So, then, when a portion of the lung is placed out of function, as it is here, and especially if it is placed out of function gradually, so that the rest of the lung may become accustomed to the inactivity of a portion of it, no apparent disturbance of respiration occurs. We have here a serous exudation, probably the result of an inflammatory process. The fact that it is unilateral speaks very much in favor of its being inflammatory. The appearance of the liquid is likewise in favor of its being an inflammatory product and not a transudation. In about four-fifths of the cases of pleuritis I have observed tuberculosis is present. We have found it present here. There is no doubt that in this case tuberculosis is the pathological factor in producing the disease. Our efforts must, therefore, be directed to the treatment of his tuberculosis, and we are so treating him. We are ignoring the pleuritis provisionally, because it produces no disturbance; because the quantity of fluid that is present is so comparatively small that we can ignore it. As I said, we are treating his tuberculosis; we are treating it with creosote. We are still in the experimental stage of our treatment of tuberculosis. We hope some day to pass out of the experimental stage into that of certainty. Until now we have not been able to.<sup>1</sup>

#### TACHYCARDIA.

At the last clinic we found that this man, who had caught a cold, as he said, four months before, had become affected with a cough

---

<sup>1</sup> Since this lecture was delivered, the certainty of the value of oxytuberculine in the treatment of tuberculosis has been established.

which continued three months, then ceased, and was followed by pain upon the right side; he had had œdema of the lower extremities, and had had a pulse varying from 150 to 165 during the entire time that he had been under observation in the hospital. We had examined him and found the area of cardiac dulness increased in the vertical diameter. We had found that the tones were very feeble but clear. Upon examining the pulse we found it still very rapid,—160. We found the tension of the pulse very low, and, as I told you, it resembled the pulse of a dying man, and the question we had to consider was, What is the reason of this tachycardia?

We may have a rapid pulse in the first place from fever. Secondly, we may have a rapid pulse from excessive heart feebleness. Thirdly, we may have a rapid pulse from paralysis of the pneumogastric nerve; and, fourthly, we may have a rapid pulse from an irritation of the sympathetic nerves. The nerves that supply the heart are the pneumogastric and the sympathetic nerves. There are fibres passing to the heart from the pneumogastric and from the sympathetic nerves. These nerve-fibres unite to form a plexus, and in this plexus they terminate in ganglionic cells, and from these ganglia fibres then pass to the muscular structure of the heart. It is probable that in these ganglia the irritation that passes to them from the pneumogastric and the sympathetic nerves are stored up, and a discharge of these ganglionic cells produces the contraction of the muscles of the heart. At least, this is one theory. Another theory is that the alternative contraction and relaxation of the muscles of the heart are the result of the inherent property of the muscle,—namely, that the rhythm of the heart is a result of the property of the muscular structure of the heart. This is, as I said, one theory. If it were true, we would have to acknowledge that there was no analogue to that property in any other muscle of the body with which we are familiar. Be the ultimate cause what it may, the results of physiological experimenting and of pathological observation are that the pneumogastric and the sympathetic nerves can affect the pulsation of the heart. If we irritate the pneumogastric nerve, we have a slow action of the heart. If we irritate the sympathetic nerve, we have increased rapidity in the action of the heart. Now, we said that this increased rapidity of the heart's action that we have observed here might be due to fever. Well, our patient had no fever; so we can exclude that. There, therefore, remain

for us either an affection of the heart itself, or an affection of the nervous tissues of the heart, or those nerves which supply the heart. Is there anything in this case to suggest the probability of the trouble being in the heart itself, or, we will say, in the muscles of the heart? The œdema that this man had proves beyond possibility of doubt that there must have been some enfeeblement of the heart; that the pumping apparatus was not doing its work; that there was a venous stasis causing the œdema. Now, is there anything that confirms this opinion from the history? He had dyspnœa upon exertion. Now, this dyspnœa upon exertion could be due to some functional derangement, or to some affection of the heart. We find no trouble in the lung. We found nothing that could explain the shortness of breath except this enfeeblement of the heart, and, taking these two facts together,—the œdema of the lower extremities and the shortness of breath upon exertion,—we know that there must be an enfeeblement of the heart present. You remember that the pulse was of very low tension. You remember that when you put your finger upon it you could suppress it with very slight pressure; so that you know that, although from lying in bed and keeping quiet, the œdema had disappeared, yet there was still present, although to a less degree, feebleness of the heart that had produced the œdema of the lower extremities. But then you found an increase in the area of cardiac dulness in the vertical diameter; that shows an increase in volume of the left ventricle. Now, an increase in volume of the left ventricle could be due either to an hypertrophy or to a dilatation. If it were due to an hypertrophy, you would have a strong, full pulse, provided there was no valvular lesion present. If you had an hypertrophy of the left ventricle without a valvular lesion, you would have a firm, vigorous pulse. You would hear an accentuation of the second aortic tones, and you would hear loud tones at the apex, but, as a matter of fact, we had a feeble pulse, and we had feeble tones, both at the apex and the aorta, showing that there could not be an hypertrophy, an increase of the muscular tissue of the right ventricle, present, but that there was—there must be—a dilatation of the left ventricle. A dilatation of the left ventricle occurs whenever the muscular tissue of the left ventricle fails to act with sufficient force to counteract the distending influence of the blood which is forced into it; it expands, it dilates. This will occur when the amount of work which the heart has to

overcome is greater than the muscle can perform. Supposing we have a valvular lesion. Let us say we have an insufficiency of the aortic orifice. Then the left ventricle would become filled from both sides, from the aorta and from the left auricle, and the consequence would be possibly an over-stretching of the tissue of the heart, and, in consequence of this over-stretching, an enfeeblement of the muscle, and, finally, a permanent distention. Or, if there was some other obstruction to the onward flow of the blood, there could, in the same manner, be an incomplete emptying of the ventricle during systole, and a portion of the blood remaining behind in the ventricle would be added to that blood which would be forced into the ventricle from the auricle, and again an over-stretching of the ventricle might occur. Such might be the case during severe exertion. An individual whose heart was not perfectly normal, the muscular structure of whose heart had become enfeebled, either from lack of proper nutrition, or from overwork, or from the use of alcohol, or from any cause whatsoever, might, in consequence of severe physical exertion, produce an over-distention of the left ventricle and a weakening of that tissue, producing a permanent dilatation. Our patient tells us that before he took sick he worked seventeen hours a day, and that a portion of this work was hard, physical work; that he had lifted rather heavy weights. It is probable that this physical exertion, long continued, produced an enfeeblement of the heart, and the final result of that enfeeblement of the heart was that it finally gave way suddenly. You will remember that our patient told us that he was a heavy drinker and smoker, and you remember that, on former occasions, we spoke of the deleterious influence of tobacco and alcohol upon the heart. Possibly if this man had not been in the habit of abusing himself with these two poisons he might have performed the work that he did without any injury ensuing.

We spoke of the nerves of the heart, and we said that the increased frequency of the heart's action might be produced by a paralysis of the pneumogastric nerve, or by an irritation of the sympathetic. Now, these lesions are rather obscure. It is very difficult to diagnose either the one or the other, and we can only do so with absolute certainty when we have other signs than those from the heart to make the diagnosis complete. It is an accepted fact that exophthalmic goitre is an affection of the sympathetic



nerve. The symptoms of exophthalmic goitre are goitre, increased frequency of the heart, exophthalmos, and tremor. These are the four prominent symptoms in exophthalmic goitre. Now, either one of these symptoms may be absent. We may have this disease, which is called Graves's disease, or Basedow's disease, in an undeveloped form without the exophthalmia, or without the goitre, and in such case we might have increased frequency of the heart's action that would be due to an affection of the sympathetic nerve, and yet there might be some difficulty in recognizing the trouble. Now, our patient, as I say, has no exophthalmia, has no goitre, and you see, when he extends his hand, that there is no trace of tremor; so we can exclude exophthalmic goitre. We can exclude Basedow's disease entirely. There is no other symptom that I have found that would direct our attention to the nervous system. But, gentlemen, we have another argument, which is not only the strongest that we can use, but likewise the most agreeable to employ,—namely, we have cured the condition. At the last clinic we ordered this man digitalis; he was given a fluid extract of digitalis with the elixir of calisaya, in the dose of three minims of the fluid extract to a half-ounce of the elixir. He has taken three of these doses daily since Saturday; in other words, he has taken, up to the present time, seven doses; he has taken twenty-one minims of the fluid extract of digitalis, and if you will now feel his pulse, you will see that the whole picture has been changed. He now has a pulse of 84, comparatively strong and slow, and the sphygmogram which I here show you indicates the change. You remember the appearance of the sphygmogram at the last clinic. You see the tracing which I have just taken. This tracing, compared to the one taken on Friday, shows the pressure is very high; compared to the normal, the pressure is very low.

Upon percussion we find now the upper border of the area of cardiac dulness at the lower border of the third rib, the right border at the left border of the sternum, and the apex beat at the fifth rib, about one centimetre to the right of the mammary line. The apex-beat, you remember, was somewhat lower on last Friday; it was in the fifth intercostal space in the mammary line about two centimetres lower and one centimetre farther to the left than it is to-day. That indicates that the left ventricle is not as large as it was three days ago, that it contracts better, that it has overcome the distention that was present, and that it is more nearly normal than it was at

that time. You hear the tones clear, but still feeble; far louder, and stronger than they were three days ago, but very much feebler than is normally the case. So, then, our case has become a clear one. *Pari passu* with the reduction in the pulse, there has occurred a reduction in the size of the heart, and at the same time an increase in the force of the tones. The pulse is still somewhat rapid; in a man of fifty-eight, it should be about seventy in the horizontal position. The pulse is still feebler than normally. The patient has not been entirely cured of the trouble in the heart, but he has been immensely improved, and you see his appearance has improved at the same time. His color is better, the expression of his face is a more pleasant one, and he tells us that he feels a great deal better. There is still one point that we have not explained. This patient tells us that he has pain; has had pain for the last month upon the right side. That may be due to congestion of the liver; it may be due to pleuritis, or to intercostal neuralgia. Let us see whether it is or not. Unless we explain every symptom that the patient has, we have failed to do our duty as clinicians. It will not do to pick out one symptom, explain that, and then leave the case. The little symptoms that you do not think of sufficient importance to investigate may be of the utmost importance,—may really change the whole complexion of the case,—and until you have explained all of the symptoms that are present in any case you have not fully done your duty, either to yourself or to your patient. Now, we will press the intercostal spaces, and, as you observe, the patient complains of pain upon pressure upon the right side at the lower intercostal spaces. All the intercostal spaces, from the seventh down, upon the right side are sensitive. You see that there is a very marked difference between the sensibility upon the left side and that upon the right. And if I percuss upon the vertebral column from above downward, the moment that I reach the sixth dorsal vertebra he indicates by squirming that the region is sensitive. From the sixth dorsal vertebra down to the eighth there is decided sensibility. The pain that this man has, then, is intercostal neuralgia. We will relieve him of this, in all probability, by applying a blister to the sensitive vertebra.

As to the digitalis, we would advise that it be continued, but it will require watching. As soon as you get the effect of digitalis, be careful. As soon as you find that your digitalis has begun to act, then it is time for you to be on your guard. Watch the pulse care-

fully. If you don't, if you keep on giving your digitalis for a long period of time, you will some day find your patient suddenly poisoned with that drug. For, as you probably know from your lectures upon *materia medica*, digitalis is a drug that is cumulative in its action, as if it were stored up in the body, and then suddenly acted. As soon as the pulse reaches 70 we will stop with the digitalis; until then we will continue to give it as we have done. We will have the patient lie quietly in bed, as he has been doing, and, in all probability, he will become as well as he was before the disease attacked him.

# Neurology.

---

## SPINAL IRRITATION.

BY T. McCALL ANDERSON, M.D.,

Professor of Clinical Medicine in the University of Glasgow; Physician to the Western Infirmary, etc., Glasgow, Scotland.

---

In the year 1828 the late Dr. Brown, of Glasgow,<sup>1</sup> directed attention to a class of cases illustrative of disorder of the spinal cord, to which he gave the name of spinal irritation (now sometimes called rachialgia). This affection had previously been alluded to by Mr. Player, of Malmesbury, in an article in the *Quarterly Journal of Science* for January, 1822, and a good many years afterwards it formed the subject of important contributions to our knowledge of it as a distinct affection from the pen of the late Mr. Teale, of Leeds,<sup>2</sup> and a few years later from the Messrs. Griffin, of Limerick.<sup>3</sup> To these gentlemen we owe almost all that we know of it at the present day.

It is specially apt to occur in debilitated, nervous, and hysterical subjects, and, although it is sometimes met with in males, it is, *par excellence*, a disease of the female sex. This is well shown by the statistics of the Messrs. Griffin, for of one hundred and forty-eight cases, twenty-six occurred in males, forty-nine in married women, and seventy-three in girls. According to Radcliffe, and this is in accord with my own experience, a strain or blow upon the back is apt to prove its starting-point, and it is the opinion of some that it is at times hereditary.

---

<sup>1</sup> "On Irritation of the Spinal Nerves." By Thomas Brown, M.D., Glasgow Medical Journal, vol. i. p. 181, 1828.

<sup>2</sup> "A Treatise on Neuralgic Diseases of the Spinal Marrow and Ganglia of the Sympathetic Nerve." By T. P. Teale. London: Highley, 1829.

<sup>3</sup> "Observation on Functional Affections of the Spinal Cord and Ganglionic System of Nerves, in which their Identity with Sympathetic, Nervous, and Irritative Diseases is illustrated." By Wm. Griffin, M.D., and David Griffin, M.R.C.S. London: Burgess & Hill, 1834.

The true nature of this morbid state is much disputed, and as the disease is one which is seldom, if ever, fatal, it is somewhat difficult to place its pathology upon a reliable basis. According to Brown, "the immediate cause . . . is spasm of one or other of the muscles, arranged along the spine, altering the position of the vertebræ, or otherwise compressing the nerves as they issue from the spinal marrow." Teale, on the other hand, attributed it to congestion, which by continuance and repetition may so far impair the tone of the capillaries as to produce a state of actual inflammation; while Radcliffe seems of opinion that the opposite condition—namely, capillary contraction and bloodlessness—is nearer the truth. But whatever the correct interpretation may be, certain it is that the abstraction of blood by leeches or cupping-glasses, applied over the tender spine, and the application of blisters in the same situation,—that is, the usual remedies for local congestion,—are the most efficacious means of cure.

The most characteristic symptom of spinal irritation is tenderness of the spine, which may implicate it in its whole length, but much more frequently at one or several parts, and the symptoms of functional derangement of internal organs and the pain so often complained of generally bear some relation to the seat of tenderness. Sometimes the part implicated is best brought out by passing a hot sponge along the spine, a feeling of scalding being experienced when the part implicated is reached. In a large proportion of cases the patient makes no complaint of uneasiness in the region of the spine, and when asked if he has any pain in the back, answers as often in the negative as in the affirmative, so that, unless this symptom is specially looked for and the spine carefully examined, the tenderness is exceedingly apt to be overlooked. For this reason, and because there is hardly a single disease in the whole category of ailments which may not be more or less accurately simulated by it, errors of diagnosis are of every-day occurrence. The following points, all of which, with the exception of the last, perhaps, I can verify from my own experience, are specially insisted upon by the Messrs. Griffin as aids to the diagnosis:

1. The pain or disorder of any particular organ being altogether out of proportion to the constitutional disturbance.

2. The complaints, whatever they may be, usually relieved by the recumbent position, always increased by lifting weights, bend-

ing, stooping, or twisting the spine; and among the poorer classes, often consequent to the labor of carrying heavy loads, as in drawing water, etc.

"3. The existence of tenderness at that part of the spine which corresponds with the disordered organ, and the increase of pain in that organ by pressure on the corresponding region of the spine.

"4. The disposition to a sudden transference of the diseased action from one organ or part to another, or the occurrence of hysterical symptoms in affections apparently acute.

"5. Perhaps we may mention the occurrence of continued fits of yawning, or sneezing. They are not very common symptoms, but, as scarcely ever occurring in acute or organic diseases, they may generally be considered as characteristic of nervous irritation."

With these preliminary remarks, I pass on to give a few illustrations of this curious complaint, commencing with the most familiar varieties.

CASE I.—A short time ago a lady brought her daughter to me from the country because she feared she had heart-disease. On inquiry it seemed that she had for a considerable period of time suffered from palpitation, and from pain immediately below the left nipple, and on asking her to point to the affected part, she covered it with the point of the finger, not with the palm; that is to say, the uneasiness was very much localized, as it so generally is in these cases. I asked her if she had any pain in the back, and she answered no; but, on examination, very decided tenderness was experienced on pressing upon the spine opposite the seat of pain in the side. She seemed in every other respect to be a very healthy girl, and I was able to assure her that those who feared they had heart-disease generally had it not, and that there was a good prospect of speedy amendment. A fly-blister was applied over the tender part of the spine, and a fortnight afterwards, when I next saw her, the spinal tenderness had completely disappeared, and the palpitation and pain below the nipple were no longer complained of.

CASE II.—The patient is a strong, robust-looking young woman, aged twenty-five, and evidently hysterical. She was admitted into the hospital on November 15, 1880. She made no mention of any affection of the spine, but complained of pain underneath both mammae, and in the left iliac region. The pain under the left mamma had been present for about eighteen months, and was of

a dull, aching nature; that on the right side was much more acute, and commenced suddenly in August last.

The thoracic and abdominal viscera seemed quite healthy, but on making firm pressure with the fingers along the spines of the vertebral column, two tender spots were discovered at different levels, corresponding very much to the points at which the spinal nerves issue which supply the areas in which pain was complained of. The patient winced visibly when those points were pressed.

A tonic mixture containing strychnine and quinine was ordered, and on November 18 two large blisters were applied over the tender parts of the spine. This was followed by a certain degree of relief from the mammary and iliac pain, by no means, however, complete. On the 29th, therefore, the blisters were repeated, and on December 8 the patient felt quite relieved of her symptoms.

CASE III.—The patient was aged twenty-two, and enjoyed fairly good health, until three months before admission to the hospital, November 27, 1880, she suffered from pain about three inches below the left nipple, which was increased on deep inspiration; she had also a dry, spasmodic cough without any expectoration, and exercise of any kind rendered her breathless. After taking food, too, she complained of pain in the gastric region, and was troubled with sour eructations. Her appetite was good. Two years ago the menses were suppressed for three months, and during that time she had an attack of pain in her breast, as at present, but on the return of the menses the pain disappeared. There was no apparent disease of the stomach at the date of admission to the hospital, and the heart and lungs were sound. On examining the spine, however, tenderness was detected from the sixth dorsal to the first lumbar vertebra, and chiefly on the left side of the spine. Of this condition the patient was quite unaware until examined. On December 10 she was wet cupped over the upper portion of the tender part of the back to ten drachms. The almost immediate result of this was the entire disappearance of the spasmodic cough and the stomach symptoms, but the mammary pain still remained. On December 16 the cupping was repeated over the lower portion of the tender part, with the result of removing the pain of the side.

CASE IV.—A. B., a rather anæmic, weakly-looking young woman, aged twenty-one, was admitted on June 2, 1879, complaining of persistent vomiting of several months' duration. She had been

exceedingly healthy until her seventeenth year, when menstruation was suppressed for two months, and since then she has always complained of more or less weakness. Her occupation as a weaver was very laborious, the hours of labor being long, and she had to work in a bent posture, the chest being almost constantly pressed against a steel bar in front of the machine. Her meals were hurriedly taken, and her diet consisted chiefly of tea and bread and butter. In January last she became unable to work, her appetite failed, and she suffered from pains in her chest; her breathing became very labored, and at times she had a sensation of choking, feeling a desire to have the window opened. She was also troubled with a hard, dry cough, unattended with expectoration. The application of mustard poultices and, later, of tincture of iodine failed to relieve the pain. About the middle of January she began to vomit mouthfuls of food, about fifteen minutes after meals. The vomiting was easy and painless, and there was no preceding nausea nor any sensation of pain while the food lay in the stomach. The vomited matters consisted of undigested food, mixed with green streaks and patches, and blood was never observed to be present. The regurgitation of the food went from bad to worse, occurring after every kind of food, and at gradually decreasing intervals after meals, sometimes even taking place during the act of eating. In March last a blister was applied over the epigastrium, and was followed, after a week or two, by iodine; but this treatment afforded no relief. In spite of everything which was tried the symptoms became gradually worse, and in May she began to suffer from severe pain across the stomach and upper part of the bowels. The pain, which was constant, and so severe as to confine her to bed, subsided shortly before admission to the hospital, at which time she was in a state of the most extreme emaciation. Since the commencement of her illness her bowels have been very costive. She now menstruates regularly. On examining the abdomen, it was seen to be extremely collapsed, so much so that normal tympanites was difficult to make out, and the abdominal aorta was seen pulsating quite distinctly. There was a certain degree of tenderness in the epigastrium, and pressure here caused some convulsive twitching of the trunk and hands. No tumor could be felt, and there was no evidence of disease in other organs. An examination of the spine, however, showed that there was distinct tenderness for about two or three inches at the junction of the



middle and lower dorsal regions. A consideration of the symptoms of the case appearing to negative the existence of any organic disease of the stomach, or any cerebral affection, it was suspected that they might be due to spinal irritation. The tenderness of the spine, age and sex of the patient, her nervous, almost hysterical temperament, and the character of the symptoms generally supported this diagnosis.

The treatment consisted in the application of a blister over the painful part of the spine. The vomiting ceased after the blister rose, and the patient has vomited only once since, and this was in connection with taking some purgative medicine. Her diet, which at first was rather restricted, has now been enlarged, and she was dismissed in perfect health.

CASE V.—Many years ago a laborer, aged forty-two, was admitted into the Royal Infirmary, complaining of pain which, commencing in the lumbar region, “went in stounds” down the outside of the right thigh and leg nearly to the ankle,—i.e., along the course of the sciatic nerve. Two years previously he had an attack of what he called lumbago, the pain of which was aggravated by motion, especially on stooping or rising suddenly, but on that occasion the leg was not implicated. The attack of sciatica commenced two months prior to admission, the pain in the limb being much more marked than in the back. It was increased by motion and by pressure behind the trochanter, and was worse at night.

No history of venereal affection of any kind could be obtained, however, although a number of small, coppery, erythematous spots, about two to four lines in diameter, were scattered over different parts of the trunk and limbs, which had rather a suspicious appearance. There was no evidence of digestive derangement; the tongue was clean, bowels regular, skin rather muddy, pulse 82, regular, and of good strength.

This was doubtless a case of sciatica, but sciatica merely means pain along the course of the sciatic nerve; and it is therefore necessary, in every case, to endeavor to ascertain the cause of the pain, or rather the nature of the condition of which the pain is the expression. In some cases no cause can be detected, and then the pain itself must be directly attacked, as, for example, by the subcutaneous injections of morphine, or the use of the continuous current.

Sciatica is sometimes the result of an injury, or is due to inflam-

mation attacking the sheath of the nerve, but there was no evidence of either of these in this case. It is sometimes a manifestation of gout or rheumatism, but the patient was apparently neither gouty nor rheumatic, nor had he any hereditary tendency thereto. It is sometimes the result of digestive derangement, but the digestive organs appeared to be in good order. It may occur in connection with gonorrhœa, in which case it must be regarded as a variety of gonorrhœal rheumatism, but the patient had no discharge from the urethra and there was no history of gonorrhœa. It is occasionally a symptom of syphilis, and in this case the dusky tint of the skin, the coppery stains, and the nocturnal exacerbations of the pain led to the suspicion that it might possibly be the result of this taint.

Finally, it might be a symptom of spinal irritation, in which case pain on pressure over the spine should be present. A careful examination of the spine was accordingly made, although the patient said that he had no uneasiness in that situation. On inspection no abnormality could be detected, but very decided tenderness on pressure was noted over the spinous processes of the upper lumbar vertebræ to the extent of about two inches. The tenderness was equally distinct on each side of the spine in this situation, but pressure upon the tender part did not aggravate the sciatic pain. Notwithstanding this, the conclusion arrived at was that in all probability the sciatica was the result of spinal irritation.

Accordingly, six leeches were applied on two occasions over the seat of the spinal tenderness. The first application diminished the spinal tenderness, while the second removed it almost entirely and diminished the sciatic pain. A few days afterwards a fly-blister was applied in the same situation, and by the time it had healed both the sciatic and the spinal pain had completely disappeared.

CASE VI.—Here is an instance of spinal irritation simulating disease of the liver. A few years ago I was hurriedly summoned to the bedside of a lady, aged about thirty, whom I found in great distress. She complained of intense pain in the hepatic region in the situation of the gall-bladder, a pain which was constantly present, but with paroxysmal aggravations. It seems that it set in gradually, and I was not called until it became intense, and was accompanied by sickness and vomiting. At first I thought that she was passing a gall-stone, and all the more so as she told me that she had a similar attack some years before. But, on inquiry, I ascer-

tained that she had no jaundice in connection with the former attack, nor was there any trace of it during this one, and that she was not aware that she had ever passed gall-stones; although little reliance can be placed on that, seeing that they are often overlooked. I also observed that she was a delicate-looking and nervous person, and that the pain was aggravated by movement. I therefore asked her if she had any pain in her back, and she said that she had. On examining it I found that, in the dorsal region, at a point corresponding to the seat of the hepatic pain, the spine was exquisitely tender. Little doubt therefore remained in my mind that it, and not the liver, was the prime offender. Accordingly a large blister was applied over the tender part of the spine. Next day there was great improvement, and in a few days thereafter she was able to be up, although the pain in the back and spine was still a little troublesome, especially when she exerted herself. A second blister was therefore applied after an interval of three or four weeks, in the same situation as the last one, and with immediate improvement, and she was soon convalescent and able to go about.

The next case is an illustration of a variety of spinal irritation which occurs more frequently than is perhaps generally supposed, for its true nature is very apt to be overlooked.

CASE VII.—On the 20th of July, 1871, I was requested to see, in consultation with Dr. Axford, of St. Leonard's, a gentleman aged about forty-five, who, with the exception of an attack of secondary syphilis about twenty-five years before, enjoyed good health, until fifteen years previous to my seeing him, at which time he had a very severe attack of gastric fever. For this stimulants were administered very freely, which seems to have proved the starting-point of intemperate habits, which continued at intervals till recently.

He never drank excessively (indeed, was usually very temperate) in society, and entertained large parties at his own house without even being suspected of inebriety; but no sooner had the last guest retired than he made his way to the dining-room, drank large quantities of any kind of stimulant which was at hand, and in a few minutes was drunk and incapable. This was repeated almost every night for months sometimes, so that the extent to which his system was saturated with alcohol may be readily appreciated.

This induced two attacks of eczema, one of them, at least, being a most aggravated one, which implicated almost the whole body, and

which lasted some months. He had likewise two attacks of what Dr. Axford described as rheumatism of the abdominal muscles. The first was treated by a German surgeon, who mistook the case for peritonitis, and treated him with mercury and leeches applied in great numbers to the abdomen, which weakened him very much; the second by Dr. Axford, and yielded in twenty-four hours to narcotics and soothing remedies.

On recovering from the second attack of eczema, at the close of 1870, he gave up the use of stimulants entirely for three months, and since then, being much debilitated, and his strength not returning, he resorted to them in great moderation and under medical advice.

When I saw him, with Dr. Axford, he was very weak, could take very little exercise, and had great difficulty in walking up-stairs or in going up-hill, so much so that his friends feared that paralysis was setting in. His legs were slightly œdematous, and his pulse, which was rather weak, was regular though rapid, being rarely under 100 per minute.

His skin was very dark and swarthy, especially that covering the legs, where it was almost coppery, so much so that Dr. Axford, viewing this circumstance along with his extreme debility, feared the onset of suprarenal disease.

He slept well, and his appetite was good; but for a good many weeks he ate very little, owing to a frequent obstruction to the passage of food into the stomach, accompanied by severe pain, which he referred to the lower third of the sternum, and which was evidently due to spasm of the œsophagus. This spasm only occurred at times, and especially when hard or hot food was swallowed, and sometimes it lasted several minutes, and yielded pretty suddenly, when he felt the food "fall into the stomach." If he swallowed a mouthful of fluid the spasm was occasionally overcome, but if not, it increased the obstruction and intensified the pain. Physical examination of the throat and chest yielded negative results, but marked tenderness of the spine was detected in the upper dorsal region to the extent of several inches, but more at some points than at others.

The urine was free from albumen, but frequently contained crystals of uric acid, which were passed without pain. The bowels were regular.

Many of the symptoms presented by this patient were of a nature to cause much anxiety as to the future. The dusky tint of skin and the debility pointed somewhat to Addison's disease, but it was hoped that the former was the result of by-gone attacks of eczema, either alone or in conjunction with the old-standing syphilitic taint, which is well known to favor deep pigmentation of the skin, and that the latter was sufficiently accounted for by the previous illnesses and, above all, by the intemperate habits.

The history of the case led us to fear that structural changes might have commenced in the liver; but there were no symptoms pointing with any probability to that conclusion.

The spasmodic affection of the œsophagus made us suspicious of the presence of an intrathoracic tumor, syphilitic, aneurismal, or otherwise; but apart from the spasms there was no evidence whatever of such a complication. On the other hand, there was no indication of permanent œsophageal obstruction, as the food was only at times checked in its passage downward, although too much stress must not be laid upon this fact, for it is well known that spasm of the œsophagus, as of other parts, may occur as the result of tumors which have no direct connection with the œsophagus, and which hardly press upon it at all. But the very decided tenderness of the spine led us to hope that that was the cause of the spasms, and that its removal, coupled with the use of means to soothe the nervous system and improve the general health, might have the effect of dissipating it. The prognosis, therefore, depended a great deal upon the result of the treatment, which was, therefore, looked forward to with much interest.

A long, narrow blister was applied over the tender part of the spine; light nourishing diet, two glasses of sherry per day, and the following mixture were prescribed:

R Potassii bromidi,  $\mathfrak{z}\text{ii}$ ;  
Potassæ bicarbonatis,  $\mathfrak{z}\text{i}$ ;  
Carb. ferri saccharatæ,  $\mathfrak{z}\text{ss}$ ;  
Inf. calumbæ, q. s. ad  $\mathfrak{z}\text{iii}$ .

Sig.—A tablespoonful in a wineglassful of water thrice daily.

In a letter received from this gentleman's wife five days after my visit, she wrote, "The spot in the back which you found tender, and over which the blister was applied, has become very painful, so much so that he feels it whenever he moves or coughs, a thing which he

never experienced before. He took food both yesterday and to-day without any choking, the first time for many a month." After the first blister was healed a second one was applied, and in a short time thereafter the dysphagia finally disappeared. That these applications were the curative agents is proved not only by the immediate improvement in the power of swallowing, but also by the increased sensitiveness of the spine immediately succeeding the application of the first blister; while the disappearance of the dysphagia, coincident with removal of the spinal tenderness, showed that the former was one of the manifold symptoms of spinal irritation.

The following case is also worthy of being recorded.<sup>1</sup>

CASE VIII.—E. L., aged twenty, a servant, was admitted to the Western Infirmary on February 11, 1897, complaining of head symptoms, with spasm of the eyelid and lower jaw, of one year's duration.

*Family History.*—Her father, aged fifty-six, has been for four years confined in a lunatic asylum, loss of memory being the only marked symptom of his illness which his wife remembers. A sister of his was also confined in an asylum, where she died at the age of twenty-six. The mother is alive and well, and there is no nervous inheritance on her side of the family. Of the four children, a boy and a girl died in infancy, the third, a girl, had an attack of chorea at the age of sixteen, and the fourth is the patient herself.

*Personal History.*—The first illness from which she is noted to have suffered was enlargement of the glands on the right side of the neck, when she was seven years old. This decreased as she grew older, although it is not entirely absent. She began to menstruate at fourteen, was always regular, and remained healthy till a year ago. She never had fits, either epileptic or hysterical, and no exciting cause for her illness can be ascertained. In particular, there is no history of shock or fright. Her mother stated that she had always been somewhat excitable.

*Present Illness.*—A year ago she began to suffer from severe attacks of neuralgia, limited to the right temple, and during these there was often paræsthesia, there being a sensation as of water running down the cheek. A month afterwards there followed uncontrollable spastic movements of the lower jaw, associated with a click-

---

<sup>1</sup> Reported by Dr. William R. Jack.

ing noise produced in the mouth by the tongue. She had also a "heavy feeling of weakness" over the vertex cranii, and there was spasm of the eyelids. These symptoms occurred on four consecutive nights, about 8 P.M., and lasted about fifteen minutes. A month later there were trifling repetitions of the clicking sound during the day, soon followed by spastic attacks, which sometimes continued all night, and sometimes passed off in a few minutes, to reappear next day. Since then the symptoms have remained substantially the same, save that the attacks have increased in frequency. She knows when one is about to come on by a feeling of stiffness in the eyelids. Five minutes later, clonic contractions of the lower jaw, above referred to, set in, and, along with these, rapid clonic contractions of the eyelids, ending in a tonic spasm, when the orbicularis resists attempts to open the eyes. Each attack lasts about ten minutes. During its continuance she cannot speak or open the eyes, but understands clearly everything that is said, and says she hears more acutely than usual. At these times she often notices a peculiar smell, as of boiled cabbage-leaves, and has a great aversion to noise. For the last year, a day has rarely passed without a renewal of the symptoms in a more or less severe form. When the attack is mild, the eyes remain open. Sometimes there is an excessive flow of saliva, which dribbles from the corner of the mouth. In her sleep she is restless, moving her hands and legs about. She is of an emotional nature, sometimes weeping without apparent cause. There is never unconsciousness, nor has the tongue been bitten. She has control over the bowels and bladder. Her appetite is good, and there is no constipation.

Owing to her mistress's indulgence, she retained her situation until three months ago, when she gave it up. While in service she managed to perform her duties, and could carry plates, etc., without breakage, so that there would appear to have been no spastic movements of the arms.

*Present Condition.*—The girl is slimly built, nervous in appearance, and of a sallow complexion. There is an enlarged glandular mass below the right side of the jaw. The pulse is somewhat rapid (96), but regular. The lungs, heart, liver, and abdominal cavity present no abnormality. The urine is normal. Temperature 98.4° F.

The pupils are equal, and respond to light and accommodation. There is no nystagmus.

There is distinct tremor of the legs as she lies in bed, especially when an attack begins. It is not greater on one side than on the other.

Both knee-jerks are exaggerated, and there is a tendency to ankle-clonus, especially on the left side. The forearm reflexes are somewhat more evident than usual on both sides. Even a slight touch is readily perceived on both sides, but with the *æsthesiometer* it is found that on the legs the two points are felt as one at their farthest separation, and on the left arm only when they are farther apart than on the right. The sensibility of the finger-tips appears unchanged. There is no thermo-anæsthesia, and no loss of the sense of pain or of the muscular sense.

Pressure over the points of emergence of the supraorbital, facial, and other nerves in the face reveals no tenderness. Pressure over the various "hysterogenic zones" on the anterior surface of the body does not give rise to tenderness or develop an attack. There is, especially, no ovarian tenderness on either side, and pressure in those regions does not affect the symptoms. Over the cervical spine, however, a tender area is found, extending from the third to the sixth cervical vertebra. In this region pressure is followed by the onset of an attack.

On March 12 a fly-blister was applied over the tender portion of the cervical spine, after which the spasms entirely ceased for four days, but then returned slightly, so that the blister was repeated on March 22, and again on April 8, and with complete success.

On April 3 a mixture was prescribed containing potassium bromide and tincture of *nux vomica*, and on May 14 the patient left for the Convalescent Home much improved in every respect.

But the most remarkable instance of this complaint which it has been my lot to encounter came under my notice while physician to the Royal Infirmary in 1871.

CASE IX.—This patient, aged seventeen, a plumber, was admitted on June 1.

It was said that he had been very nervous all his life, but had otherwise enjoyed good health until about three years prior to admission, when one forenoon, while at Ayr, he felt very sick, and vomited, and his abdomen swelled,—symptoms which were some-



what relieved by an enema. On that day, too, his hands and his head shook for a short time at intervals, and he complained of severe palpitation. For two and a half months he was unable to work, and seems to have had one or more shaking fits each day, and more aggravated ones at night. For the next six months he was free of these, and was able to do light work, but complained of weakness. About this time he seems to have had some sort of tumor in the region of the upper maxilla, which was seen by one of the surgeons to the outdoor department of the hospital. It was painted with iodine, and subsided in great measure, but no sooner was it better than the fits recurred; he has never been more than eight days without one, and for the last two and a half months they have been getting gradually worse. He can generally tell when one is impending, as he has, for a minute or so before it, a "feeling of weakness," and trembling in his inside.

On entering the ward on June 2 I found him in the midst of one: he was lying on his back quite conscious, able to answer questions which were put to him and to take food, but he was flapping his arms slowly and regularly, as if they were wings, closing and opening his eyelids synchronously with the movement of the arms. If we agitated him, by proposing to interfere with these movements, for example, they became incredibly rapid. When one arm was held sufficiently firm to stop its movements the side-to-side movement of the other ceased, but he immediately began to slap the bed with it with great violence and rapidity. When both arms were bound down, he at once began to flex and extend the lower extremities with similar force and celerity. When both his arms and legs were bound down, he rocked his head from side to side with exceeding rapidity, and said he felt as if his "heart would burst." When the pressure was removed from the upper extremities the movements of the lower ceased, and those of the former recommenced.

On entering the ward on June 3, I found him in the midst of another fit, which at first presented the same characters as on the preceding day, but within a couple of minutes all movements ceased, and the muscles of the trunk became perfectly rigid. While the rigidity continued he was noticed to open his mouth, and thereupon he commenced to open and shut it with great rapidity. About a minute afterwards these movements ceased, his mouth remained widely open, and then he proceeded alternately to protrude and re-

tract his tongue with a rapidity which was perfectly marvellous. In a few minutes all the symptoms passed off, and he expressed himself as feeling well, but much exhausted. He then shook hands with me, and evidently felt much relieved that the paroxysm was over.

On the day of his admission he was put fully under the influence of chloroform, but whenever its effects passed off the paroxysms recommenced. Subsequently twenty-five grains of chloral were administered, half an hour after which he fell asleep, but woke in a paroxysm in six hours. The subcutaneous injection of one-third of a grain of morphine had a similar effect. It was thus proved that the fits, which at this time were very numerous and severe, were only temporarily relieved by sedatives and narcotics.

On careful examination of the patient on the 3d of June, it was found that the lower portion of the spinal column, from the middle of the dorsal region downward, was decidedly tender upon pressure, especially at the middle of the upper and lower thirds of this part. He was fairly nourished, but looked rather weakly and dwarfed. There was no evidence of fever; his tongue was clean, his appetite deficient, his bowels rather costive, and he denied masturbation. He was ordered light nourishing food, and two ounces of brandy in the twenty-four hours. Six ounces of blood were withdrawn by cupping from the tender spine, and a dose of chloral was repeated at night. On the 5th of June the following report was made: "Spinal tenderness all but gone; has only had a few slight fits since the cupping, and none at all since noon yesterday."

By the 7th there had been no return of the fits, but as the spinal tenderness had not entirely disappeared, a long narrow blister was applied to that region.

On the 8th of June he had one slight fit at 1 P.M. which lasted about five minutes.

On the 10th of June the following mixture was prescribed:

R Vini ferri, ℥ii;  
 Solutionis Fowleri, ℥ii;  
 Syrupi simplicis, ℥i;  
 Tincturæ calumbæ, q. s. ad ℥vi. M.

Sig.—A teaspoonful in water three times a day after food.

On the 14th of June, having been allowed to go about the ward on the previous two days, he had a severe fit, which lasted from 4.30 till 6.30 A.M., and which was followed by several slighter ones.

This is quite in accordance with what we generally notice, that the symptoms of spinal irritation are relieved by a rest and aggravated by exertion.

On the 3d of July, having had no fits since the 14th of June, and being otherwise well, although his intellectual powers were, as they had all along been, decidedly below par, he was dismissed.

Although cases presenting some features in common with the above have from time to time been observed and recorded, this is, taken as a whole, unique as far as my reading and experience go. The occurrence of anomalous functional disorders associated with well-marked spinal tenderness, and the removal of the symptoms by treatment applied over the seat of the tenderness, prove, in my opinion, the correctness of the diagnosis. It is true that a few fits—one of them a severe one—occurred after the leeching and blistering; but this by no means invalidates the conclusion arrived at, for we often find that the immediate effect of the most appropriate treatment, in this as in many other diseases, is rather to aggravate than to alleviate the symptoms, while the ultimate result is all that can be desired.

The results obtained in the preceding cases by blistering and the local withdrawal of blood were so satisfactory that it was not necessary to have recourse to other methods of treatment, such as the passage of a continuous current of electricity through the vertebral column, for which great success has been claimed.

Did space permit, many other illustrations of this curious complaint could be given. But enough has been said to prove, first, that there is such a disease as spinal irritation; secondly, that its symptoms are of the most varied kind, so much so that it may simulate almost every known ailment; thirdly, that, if we are on our guard, and make a careful examination of the spine, their true nature can generally be ascertained; and, fourthly, that remedies applied directly to the seat of the spinal tenderness, especially leeches and blisters, are the most efficient means of cure, and that, in the majority of cases, they are speedily, and often permanently, successful.

# GENERAL PARALYSIS AS MET WITH IN HOSPITAL PRACTICE.

CLINICAL LECTURE DELIVERED AT THE EDINBURGH ROYAL INFIRMARY.

BY ALEXANDER JAMES, M.D., F.R.C.P. (Edin.),  
Physician to the Edinburgh Royal Infirmary, Edinburgh, Scotland.

---

GENTLEMEN,—If I were to ask any of you to enumerate the symptoms of general paralysis, I feel sure that, whilst you would refer to the peculiar speech disturbances, the quivering of the lips and tongue, to the changes connected with the pupil, and with locomotion and equilibration, you would certainly not fail to mention the peculiar mental change which occurs, and which is so prone to manifest itself in exalted or grandiose delusions. In a typical case, so called, you would be correct, for in such a case the malady, we are taught, tends to make its commencement known by such delusions.

But you all know enough of medicine to realize that typical cases are apt to exist in the minds of the physician or student rather than in reality. You have realized that disease is not an entity, a something foreign to the organism which lays hold of it, and which bends or twists or breaks it according to its own will. Rather is it the case that disease is a condition in which one or other of the numerous and complex processes going on in the organism are interfered with or disturbed. Inasmuch as no two human beings are exactly alike, it follows that the indications of the disturbances of disease in this or that process must correspondingly vary. In this way we may say that a typical example of a disease is as unusual as is a typical example of a human being.

Let us take, for example, even such a distinct and characteristic disease as syphilis. We all know that syphilis is apt to present its symptoms in three sets, the primary, secondary, and tertiary. How differently, however, these manifest themselves in different individuals! Thus, in one patient primary and secondary symptoms may be well marked, in another only the tertiary ones may indicate the

disease. In this patient there will be skin eruptions, in that one an iritis; in this a cerebral gumma, in that a specific thrombosis; here an aneurism, there a waxy liver; here locomotor ataxy, there a transverse myelitis. All this shows, therefore, that the phenomena of disease manifest themselves differently in different individuals. Why this should be so is a very difficult question to answer; but we content ourselves by saying that it must be the result of different individual constitutional conditions and surroundings.

Let us now apply these considerations to general paralysis. This disease is best regarded as being due to an exhaustion, more or less premature, of nerve tissue, and especially of those parts of the nervous system which are concerned in the performance of the highest kind of nerve work. These parts we believe to be mainly the gray matter of the cerebral convolutions,—that is to say, the parts concerned in perception, reason, and the higher and more complex sensory motor functions.

Next observe that every human being is, as regards each of his tissues, endowed at birth with a store of vital energy sufficient to keep him living for a certain number of years. Suppose now that in an individual there is, as the result of overstrain, or excess, or injury, a too rapid using up of the parts of his nervous system concerned in perception, reason, and the higher sensory motor functions, what will be the result? This must necessarily be an impairment in the performance of these higher functions, associated objectively with a wasting and shrinking of the cerebral convolutions.

Now observe that among individual men the effects of excessive wear and tear do not show themselves in precisely the same way. This can be illustrated very well by what we notice of the effects of alcohol on brain function. We know that the general effect of alcohol is first to excite and secondly to depress; but we know that these effects manifest themselves very differently in different individuals. In one individual, for example, we find that that part of his cortex concerned in speech and ideation may continue to work vigorously, whilst that concerned in the sensory motor or equilibration mechanism cannot functionate at all. In another we find that equilibration may be hardly interfered with, whilst speech is impossible, and many other variations in the effects of alcohol can be recognized as due to individual conditions. In the end, of course, there is an absolute similarity,—namely, complete failure of all nerve functions.

So it is with general paralysis. In some cases, at first and for a time, the mental disorders are predominant, the sensory motor disturbances being less marked, whilst in others the motor or sensory motor are predominant, and the mental less marked.

But now, in conclusion to this preamble, we have to remember a very important point. This is that in our conception or mental picture of general paralysis the mental disturbances, and especially what we call the exaltation disturbances, are apt to occupy a very prominent place. The result is that when a doctor is called to an early case of general paralysis which presents this symptom, the patient is at once sent, as he ought to be, to an asylum. But, on the other hand, in a case where those mental symptoms are not so prominent, and where the motor and sensory motor are, the patient very frequently finds himself sent to the infirmary, and this all the more so because many of the symptoms which he presents closely resemble those of cerebro-spinal sclerosis or neuritis. In time, and after some careful inquiry, general paralysis is diagnosed, and the patient may be sent to an asylum. But if the mental condition is such as may be tolerated at home, and if his friends prefer it, he may never be sent to an asylum at all. What usually happens in such a case is that from time to time he comes back to the infirmary for a week or two with congestive or epileptiform attacks, and returns home on their subsidence. In the end, becoming more and more enfeebled, he returns to the infirmary, and after some weeks, or a month or two, dies there.

Now it so happens that we have at present a very good example of what we are in the habit of calling the infirmary type of general paralysis, and within the last two years we have had other two equally good cases of the same kind. To these I wish now to draw your attention.

The first case is that of Samuel N., aged thirty-two years, a miner, born at Birmingham, but resident in Fife, and who was admitted on the 4th of September, 1897, complaining of shakiness and weakness of the limbs, headaches, and attacks of giddiness, causing him sometimes to fall down. He states that he has only been ill for about six weeks, but as the patient was not very intelligent, and as his mind rambled frequently, we are inclined to think that he has been much longer ill than he acknowledges. Patient is a married man, and has four children. He knows nothing whatever of his parents,

but he has a brother alive and healthy, aged thirty-seven years. He states that he has never been addicted to alcohol, and has never had syphilis.

Twelve years ago, when he was working on a railway in the Soudan, he seems to have had a sunstroke, which laid him up for three days, but from which he recovered rapidly. After that time he seems to have been a sailor, but for the last five years he has worked as a striker in the pits. Four years ago he sustained an injury to his head by a stone falling upon it. This accident laid him up for a month.

His present illness, according to his own statement, began six weeks before his admission. He then began to suffer from severe headaches and giddiness. The giddy attacks were so severe that, to prevent himself falling, he had to hold on to any support. He noticed also that his legs felt weak, and were apt to give way under him, and that he had a difficulty in walking straight. He also noticed a tendency to drop whatever he was carrying in his hands. About this time also he noticed that he had difficulty in speaking. Being quite incapacitated for work he sought admission to the Infirmary.

*State on Admission.*—He is five feet six inches in height, and weighs eleven stones (one hundred and fifty-four pounds), and he has all the outward appearances of a powerful, well-developed man. His temperature is normal, and his pulse beats usually about 90 per minute.

*Alimentary System.*—The lips, teeth, and gums appear healthy. There is an indication of occasional slight dribbling of the saliva. The tongue is slightly furred. It is very tremulous, and the patient takes a little time before he can protrude it. Deglutition is normal, and appetite and digestion good. The abdominal viscera are all healthy.

With the exception of a slight quickening of the pulse, the circulatory system is normal, as also is the respiratory system.

*Integumentary System.*—There is no cedema, but the skin of the legs feels tense and somewhat brawny.

*Urinary System.*—No trouble with micturition. Urine forty ounces, acid; specific gravity, 1020; no abnormal constituents.

In connection with the reproductive system nothing abnormal can be ascertained.

*Nervous System.*—Sensory functions: Patient states that some weeks ago he noticed tingling sensations in the legs, but these are gone now. At present sensation to touch, pain, and temperature is normal. His sight is good, his color sense is normal, and there is no nystagmus. The pupils are equal, and respond readily to light and accommodation. Hearing, taste, and smell unimpaired.

Motor functions: Although the muscles of his arms and legs appear large and firm, the voluntary motor power is much impaired. The left hand is distinctly weaker than the right, and when both hands are held out, the left tends to tremble a little.

The superficial reflexes, plantar, cremasteric, abdominal, and epigastric, are all markedly increased. The deep reflexes, knee and ankle, are both increased, and there is a slight tendency to knee and ankle clonus. The organic reflexes are normal. On standing with his feet close together, and with eyes closed, there is a slight tendency to sway. He walks with rather a shuffling gait, and turning round is a little difficult. As regards his cerebral and mental functions, there is certainly distinct impairment. His intelligence is poor, and he has considerable loss of memory. His attention readily wanders from the subject in hand. His speech is slow, hesitating, and jerky, and the test words "Royal Artillery" and "British Constitution" are not well pronounced. He has no delusions or exaggerated ideas, and though the nurse informs us that he is at times somewhat irascible, he appears to be an easy-going and good-natured man.

Here, then, is an example of an early general paralysis of the kind which we find sent to an infirmary instead of to an asylum. It would have been of great use to get further information about him from his friends. This we have not been able to do, but if we did so, we should probably find, first, that his illness has been of longer duration than he himself states, and, secondly, that his actions have been showing more mental impairment than he would have us believe.

The disease with which we might most readily confuse it is disseminated sclerosis. In this disease the prominent symptoms are, as you will remember, giddiness, intention tremors, increased reflexes, and speech disturbances. But all these symptoms, as manifested by our patient, are more like what we find in general paralysis than in disseminated sclerosis. Moreover, he has no nystagmus, such as we find in disseminated sclerosis. We have concluded, therefore, that



it will turn out to be a case of general paralysis, and in this view we are corroborated by Dr. Middlemass, of the Royal Edinburgh Asylum, who has kindly seen the case with me.

Let me now, however, detail to you two somewhat similar cases which were in our wards some time ago, which we diagnosed as general paralysis, and which, on post-mortem examination, turned out to be distinctly marked cases of this disease.

William A., aged twenty-seven years, a seaman in the royal navy, was admitted to the Royal Infirmary October 7, 1891. He had been recommended to us by the surgeon at the Calton Jail, where he had been sent on account of an alleged theft. His family history is not at all satisfactory, showing a strong tendency to phthisis and nervous diseases.

His personal history is very good. He had been a healthy boy, and joined the navy from a training ship at the age of fifteen years. In 1889, when he would be about twenty-four years of age, he was an A. B., and was on board one of the ships of the East India Station. At that time he fell from the rigging, striking the hammock netting in his fall, and rebounding onto the deck, a height of about eight feet, landing on the top of his head. He lay unconscious for thirty-six hours, but recovered, and in about six weeks he went back to duty. Since then, however, his conduct seems to have altered, and, as going aloft made him giddy, he was kept to small bits of work, such as cleaning brasses, etc. He stated also that since his accident he had been liable to headaches.

In 1891 he came home on furlough, and his relatives noticed a distinct alteration in his behavior. He was irritable, and broke out occasionally into fits of rage. He never, however, showed any addiction to alcohol. When his leave was up he was transferred to a ship on the Home Station, and here he was once or twice punished for insubordination, because he refused to go aloft when ordered to do so, on the plea that it made him giddy. Finally he was charged with theft. He stated that this was because he had picked up a handkerchief belonging to one of the other men, and mislaid it, so that he could not find it again. The handkerchief had had some money rolled up in it, hence the alleged theft. He was dismissed from the service, and sent to the Calton Jail. There the prison surgeon noticed that his conduct was extraordinary, and advised his removal home or to the infirmary. He went home, but as shortly afterwards he had a slight paralytic attack, in which the left arm was specially

affected, he was sent on to the infirmary. During his stay in the infirmary, in October, 1891, he was inclined to be disorderly at night, but during the day was quiet and respectful. He was a healthy-looking, well-built, and muscular young sailor, and all his systems were healthy, except the nervous. On examining into the condition of his nervous system we found the following: Sensation to touch, pain, and temperature unimpaired. Sight good, color sense unimpaired, pupils acting normally, no nystagmus. Examination of the fundus revealed only slight congestion of the veins. Other special senses normal.

Voluntary motor power in arms and legs much impaired, especially in the legs. Cutaneous and deep reflexes in the legs markedly increased. Organic reflexes unimpaired. His gait was fairly good, but it was noticed that on turning round he did it somewhat clumsily. He had no exaltation ideas, and although mentally he was evidently below par, he was quite manageable. His speech was somewhat slurring, and when speaking occasional tremors of the lips showed themselves.

This patient lived on until April 20, 1893, and the following is a summary of his case. In February, 1892, and August, 1892, he had very severe and prolonged congestive attacks, being treated for them in the infirmary. In the intervals he remained fairly well, being cared for at home, and coming occasionally to see us at the infirmary. On the 16th of April, 1893, however, he was brought in with a specially severe congestive attack. In this he was mostly comatose, but at times he had epileptiform seizures. In this condition he remained till April 26, when the operation of trephining was determined upon. After the operation he seemed to rally a little, but he died on the 28th of April,—that is to say, four years after his accident. The following is the result of the post-mortem examination:

Skull abnormally thick, but not specially thickened at any one part. Dura practically healthy. Increased amount of arachnoid fluid with old standing thickening, especially on left side. Pia much more opaque than usual, especially over sulci. Relative atrophy of convolutions and widening of sulci. Ventricles somewhat dilated; no sign of hemorrhage and no basal disease.

The next case is that of Peter S., aged thirty-six years, a clerk, who was admitted to the infirmary on January 18, 1897, complaining of a bad memory and stuttering in his speech, and stating that

he had been ill for about eighteen months, but specially so for the last six months. At his examination, the patient was forgetful and inclined to ramble, and so for the complete history of his case we are indebted to his wife. His family history is very good, and as regards personal history, we find that he has never drank to excess, and that his surroundings at home and at work have been quite satisfactory. He never had syphilis. He seems to have been in all respects a healthy man until two years before his admission, when he had a fall on the left side of his forehead. The wound thus caused required stitching, and he was laid up for some days. He recovered from this readily enough, but about eighteen months ago his wife noticed that he was becoming irritable on slight provocation. Previously he had been a quiet and easy-tempered man, but now he would become noisy at times, and sometimes would finish up by crying. She told us that he did not lose interest in his work, but that, on the contrary, he appeared to be rather more anxious about it. At times she noticed that he did what she calls "queer things," he would laugh without any apparent cause. He never had any extravagant or exalted ideas while at home. On admission, however, it is noteworthy that he talked of his having been abroad, at Lucknow, Aden, and other places. He had never been there, but it transpired that his brother had been. His wife states that his gait has altered somewhat. She describes it as being more tottering than it was. About six months ago she noticed that he had some difficulty in bringing out his words, and that he would give things the wrong name without noticing his mistake. Lately his memory has been failing, so that he had to give up his situation, because he was not able to address people properly. A doctor was called in, who advised the patient to come to the infirmary, where he was admitted as above.

On admission he was found to be a fairly healthy-looking man, five feet six inches in height, and weighing about ten stones (one hundred and forty pounds).

On examination we found that, though he had manifested no symptoms of heart trouble, he had a double aortic valvular lesion with some enlargement of the heart. With this exception, and with the exception of his nervous system, he presented no other signs of disease.

*Nervous System.*—Sensibility to touch, pain, and temperature unaffected. His sight is good; there is no nystagmus. The right

pupil is rather the smaller of the two, but both react to light and accommodation. The other special senses are unimpaired. Voluntary motor power in arms and legs is slightly impaired. The cutaneous reflexes are fairly normal, but the knee- and ankle-jerks are increased. Ankle clonus is present on both sides, more marked on the left. Organic reflexes, bladder and rectum, unaffected. His gait appears slightly spastic in character, but with the feet together and the eyes closed he stands quite steadily. His intelligence and memory are impaired, his speech is somewhat slow and slurring, very evident when he tries to say "Royal Artillery." His tongue is slightly tremulous when protruded. The following is a summary of the progress in this case:

He improved considerably during his stay in hospital, and was always quiet and manageable. The nurse told me that he was always most obliging, doing everything he could to help in the work of the ward. He was sent to the Convalescent House on the 12th of March, 1897. After remaining there for three weeks, he returned home, where he remained till May 14, 1897, when, as he was more feeble mentally and bodily, he was brought back to the infirmary. He then gradually became worse, becoming somnolent and comatose. On May 26 marked Cheyne-Stokes breathing was observed, and he died on May 27, 1897, having been ill for about two and a half years. The following is the result of the post-mortem examination:

The pia and arachnoid over the vertex were greatly thickened, showing marked opacity. The convolutions were atrophied and flattened; the lateral ventricles were greatly distended. The brain weighed three pounds four ounces.

These, then, are also examples of the type of general paralysis which we often meet with in the infirmary.

The morbid anatomical changes found in this disease are numerous, and comprise thickening of the calvarium, thickening of the membranes,—dura, arachnoid, and pia,—atrophy of the convolutions, with, histologically, disappearance of nerve-cells and proliferation of neuroglia tissue,—effusion under the membranes and effusion also into the ventricles, which are usually somewhat dilated. It will be noticed that in neither of our two cases was the dura thickened, but in both the thickening and opacity of the arachnoid and pia and the atrophy of the convolutions were well marked. In

the first case also there was marked increase in the thickness and density of the bony vault of the skull.

Pathologically, in general paralysis the primary process is probably failure in the trophic power of the cortical cells, and consequent atrophy of the convolutions. To these the other changes simply follow, just as, in senile contracting kidney, failure in the trophic power of the secreting tissue of the kidney is followed by thickening of the capsule and general fibrosis of the organ.

As to why those three cases should present differences from the ordinary examples of general paralysis we can only surmise. That it may be explained by differences in the constitutional conditions and surroundings, there is every ground for supposing. In this connection, the points which seem most noteworthy are (1) that in none of them was there a history of syphilis, alcohol, or other excesses; (2) in all of them there was a history of injury to the head; and (3) in at least one of them there was a very bad family history. The relative absence of grandiose ideas and delusions and the relatively marked motor troubles may possibly be ascribed to their occupations having been such as entailed physical rather than intellectual strain. In the first two cases, the miner and the sailor, grandiose ideas were not present at all. In the third case, the clerk, they were present only to a slight extent. It would be interesting to look into the subject of the mental symptoms in general paralysis when manifested by physically working individuals, and to compare these with the changes occurring in this disease among the mentally working. I do not know, however, at present of any investigations on this subject.

Having realized that general paralysis is due to failure of trophic power, we can understand that its treatment is the opposite of satisfactory. As might be expected, we find that the rest, good diet, and quiet routine of hospital life do good in themselves. We have tried cerebrin, didymin, and thyroid extract, but without practical result. A combination of the iodide and bromide of potassium seems to do good, and for the headaches and epileptiform attacks, we have found nitro-glycerine very useful. There is every probability that trephining can exercise a prolonging effect on the disease. One or two openings in the calvarium cannot fail, therefore, to have a beneficial effect by reducing the intracranial pressure.

# Surgery.

---

## UMBILICAL HERNIA; REMOVAL OF A LEFT-SIDED GOITRE; CIRCULAR CRANIECTOMY FOR MICRO-CEPHALY; TUBERCULAR ABSCESS OF THE HIP-JOINT.

CLINICAL LECTURE DELIVERED AT ST. MARY'S HOSPITAL.

BY H. O. WALKER, M.D.,

Professor of Rectal Surgery, Genito-Urinary Diseases, and Clinical Surgery in the Detroit College of Medicine, Detroit, Michigan.

---

GENTLEMEN,—At this, our first clinic of the college term, I have a number of interesting cases to show you.

CASE I.—Mrs. L., aged forty-three, gives a history of a mild form of asthma for several years, and during one of her coughing fits about seven years ago she was taken with a sudden pain at the umbilicus, which lasted only a short time. Shortly after this she noticed a small protrusion, which has gradually increased to the size which you now see. (Fig. 1.) Her abdomen is pendulous and very obese. The protrusion is partially reducible and undoubtedly adherent. As she has never had any evidence of strangulation, it is quite probable that the sac contains omentum.

Umbilical hernia in adults is very different from that in children, and does not promise very much in the way of mechanical treatment. It is rarely necessary to operate in children, as the hernia is almost always reducible, and can be easily cured in a few months by the proper application of a truss. In adults the pathological conditions as a rule prevent complete reduction. You will observe, as already stated, that the adhesions are such that I am able to effect only a partial reduction in this case.

Not infrequently we have an addition to the true umbilical hernia by an extra protrusion of an intestine in close proximity,

known as the "para-umbilical." Such a one I operated upon yesterday for Professor J. E. Clark, in which existed a hernia not only at the umbilicus, but also just above it, which was strangulated, necessitating immediate surgical interference. This patient has worn a variety of trusses and abdominal supports without benefit. She is unable to follow her avocation, that of a cook, and for that reason has come to us for the purpose of a permanent reduction and closure. Statistics of the results of radical operations for the cure of this variety of hernia are limited, and so far as known the relapses are proportionately more frequent than in either inguinal or femoral hernia. There is good reason that this should be so, from the fact that the fasciæ and muscles in close proximity to the median line have become atrophied from the long pressure, and have, therefore, not the power of resistance that is found in the other forms of hernia. To secure a radical cure of any form of hernia is to so make the operation that the parts may be put in the best condition and position for approximation and healing. We will first make an elliptical incision, removing both the umbilicus and redundant skin. The incision of the sac is a matter of expediency, for if we find that it is exceedingly large and adherent, it will be best to excise it and approximate the divided peritoneum with catgut sutures, and then by the best possible method (if there is such a method) complete the operation. Let me state here that suturing the deep parts, and superficially packing with gauze and allowing it to heal by aseptic granulation, is not an ideal operation. You will observe that I have liberated all the adhesions to the peritoneum, and I can now readily return the sac and its contents into the abdominal cavity. The only point of the peritoneum to be sutured is the opening that I made through it when removing the umbilicus. After carefully cutting away the fibrous edges of the ring, so as to fully expose the fibres of the recti muscles, I will proceed to bring them together with a continuous suture of No. 24 piano-wire in the following manner. (Fig. 2.) Commence at the lower end of the wound, upon the left side, and introduce the needle longitudinally upward about a quarter of an inch through the full thickness of the muscle, using care not to pierce the peritoneum; then crossing over superiorly to the right side, piercing the opposite muscle as before, and then back to the left, and so on, until the upper end of the wound is reached, when the needle is pushed upward out through the integument about an inch above

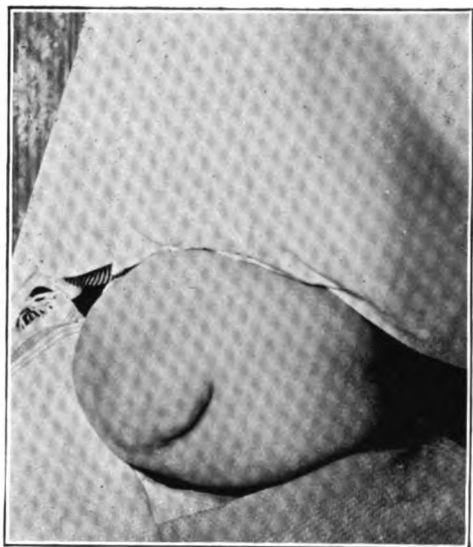


FIG. 1.—A case of umbilical hernia.



FIGS. 2 and 3.—Application of a continuous wire suture in the cure of umbilical hernia.



FIG. 4.—Final closure of the wound with silk-worm sutures.





the apex of the wound. You will notice that I have left three or four inches of the lower end of the suture free, which is threaded and the needle passed out through the integument as above. By traction at each end of the wire you will observe, further, that the edges are inverted and accurately coapted. The ends of the wire are held firmly by passing them through an inch of quarter-inch rubber tubing and winding them two or three times around it, making a sort of elastic traction. (Fig. 3.) The edges of the integument are now brought together with interrupted silkworm-gut sutures. (Fig. 4.) The object in introducing and adjusting the wire suture in the manner just described is that it may be cut off at either end and withdrawn when it is thought that the union has become firm, say at the expiration of from four to six weeks. It will be seen, therefore, that the wire is only a temporary buried suture. Buried sutures are objectionable from the fact that they are foreign bodies and are liable to produce trouble necessitating their removal at some future time, except, perhaps, in the instance of catgut, which is usually rapidly absorbed, and is therefore defective where prolonged apposition is necessary to insure firm union. I have been compelled frequently to remove buried sutures of silk, silkworm gut, silver wire, and last year I had occasion to remove a kangaroo-tendon suture that had been buried for nearly five months and had begun to produce trouble.

An ideal suture is one that can be removed after it has performed the office wanted of it. A varied experience in the use of a variety of sutures has led me to the opinion that catgut and silkworm-gut have served me the best, except in a few instances as above, when iron or silver wire has done effective work.

#### REMOVAL OF A LEFT-SIDED GOITRE.

CASE II.—Mr. E., aged fifty-three years, has had this left-sided goitre for twenty-eight years. It has not given him much inconvenience until about two years ago, when its pressure caused at times difficult breathing and a stridulous voice when talking. The usual internal and external remedies have been of no avail. Some months ago an injection of some sort was used, followed by suppuration, and there is now, as you will observe, a large pultaceous mass extruding at the point where the injection was made. (Fig. 5.) He is somewhat emaciated and has had considerable trouble of late in breathing.

I will not at this time go into the causes, symptoms, and medical treatment of goitre, but will consider it only from an operative stand-point. A great variety of methods of operation have been suggested and performed for goitre, of which but few are worthy of consideration,—namely, partial and complete resection or enucleation, and the tying of the thyroid arteries. Complete removal has been abandoned as a rule, for it has generally been followed by either tetanus or myxœdema. The dangers of this operation are hemorrhage, injury to nerves, and sepsis, the latter being especially liable to occur in this case from the fact that it is already septic. The incision that I shall first make will extend from the lower lobe of the left ear down to the sternum (S-shaped) and elliptical around the extruding mass. After dividing the integument and platysma, great care must be taken to divide all blood-vessels between ligatures.

Next comes the fascia, which is divided, exposing the sterno-cleido-mastoid and sterno-hyoid muscles. It will be necessary to sever the hyoid muscles and nick the anterior border of the sterno-cleido-mastoid, when the tumor will be fully exposed. Traction with large blunt hooks gives an opportunity for deeper inspection and division of the sterno-thyroid and sterno-hyoid muscles. The next step is the division and reflection of the gland capsule and ligating and dividing each blood-vessel as it appears in the course of the dissection. We are now enabled to pull the gland forward, and from this time on the greatest care must be exercised not to injure the recurrent laryngeal nerve or the cardiac branches of the sympathetic. In tying the inferior thyroid artery make certain it is thoroughly isolated. Our dissection is now so far advanced that we can encircle the isthmus with a stout catgut ligature and divide it. Now comes the most delicate part of this operation,—that is, the separation of the gland from the trachea, for even with the greatest care the recurrent laryngeal nerve may be injured; therefore, in order not to do so, I will transfix and doubly ligate the tumor just above its attachment to the capsule, then cut it away above the point of ligature, leaving that portion with the capsule, as in this way there is less liability of injury to the nerve. Having made certain that all bleeding points have been tied and divided muscles sutured, the wound is usually closed without drainage; but in this instance I will feel better satisfied if drainage-tubes are inserted, owing to the primary sepsis.



Fig. 5.—Ulceration of a large left-sided goitre.

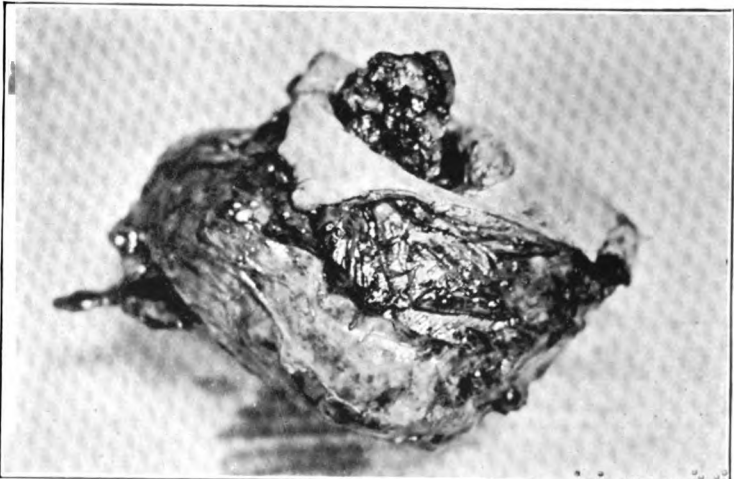


Fig. 6.—Fibrocystic goitre without evidence of malignancy.

Digitized by Google

This tumor is of the fibrous variety, and since removing it Dr. E. H. Troy has been kind enough to make a section of it with the following report: "The piece of this tumor chosen for examination was adjacent to the suppurating point, and the reason for doing so was that in the event of malignant changes taking place we would expect to find it there because of the tendency of malignant growths to break down. No malignant change found. A proliferation of epithelium which looks suspicious was observed in a few sections, but not sufficiently characteristic to be called carcinomatous." (Fig. 6.)

#### CIRCULAR CRANIECTOMY FOR MICROCEPHALY.

CASE III.—Mabel B., aged eight years; is very small for her age, with a proportionately smaller head; has a history of being fairly bright until she was nearly a year old, when she gradually developed imbecility. Her parents have no knowledge that she ever received any injury to the head, or that she ever had any severe illness. She presents now all the characteristics of an idiot,—namely, strabismus, numerous exaggerated muscular movements, involuntary discharges of secretions and excretions, frequent semi-convulsions and aphasia, making an almost constant crooning noise while awake. Your teacher on nervous diseases, Professor D. Inglis, will undoubtedly go more into details as to the causes of the interference with intracranial development than time will now permit. Suffice to say that imbecility, idiocy, and other mental defects may be due to various acute or chronic inflammatory diseases of the brain and its coverings, defective congenital brain development, neoplasms, traumatism, and extreme premature ossification of the skull. After careful observation of the child for several days, I am inclined to the opinion that we have here a case of microcephaly, and, if I am correct in my conclusions, sufficient cause for the train of symptoms that are here present,—that is, the influence of the pressure of a non-expanding skull upon an expanding brain. Lannelongue was the first to point out the possibility of benefit to be derived from surgical interference in this class of cases, since which time several others have presented papers for and against operation. However, many cases have been reported that have much benefited by craniectomies. In three cases that I have operated upon with the view of remedying a mental defect the results have been *nil*. One

in a girl of seventeen years of age, with epilepsy and great mental irritability, improved very materially for a time, but has since gradually relapsed, until now she is an inmate of an insane asylum. The other two operations were upon children idiotic from birth, of three and five years of age, without any appreciable improvement whatever. The want of improvement in these cases I have attributed somewhat to my method and to insufficient removal of the cranial vault. If we are to expect freedom of expansion of the brain, it will be necessary to remove a considerable portion of the cranial vault, or make a large osteoplastic flap of the same. Following my last operation, which occurred nearly a year ago, I determined, if ever an opportunity presented again, to do more than a linear craniectomy,—that is, make a circular ditch in the cranial vault, an idea that I considered original at the time; but I find in looking up the literature of craniectomy that Gersundy suggested a similar procedure in 1892, and it is called by him circular craniotomy, which exemplifies the familiar saying, "Nothing new under the sun."

Yesterday the child's head was shaved, washed, and a soap poultice applied and retained with a bichloride gauze cap. Before proceeding with the operation, we shall control hemorrhage as much as possible by applying rubber tubing around the head, holding it in place by four sutures, one behind and below the occipital protuberance, the second in front of the superciliary ridge, and the others just above each ear, which fix it so it cannot slip, and is well out of the way of the field of the operation. Two elliptical incisions are now made through the scalp, with the convexity of each below the parietal eminence, leaving an isthmus in front of the coronal suture and one behind the lambdoid suture. (Fig. 7.) The scalp with the periosteum is reflected upward, and we are now ready for the removal of the wide strip of bone. I usually make a starting-point with a small trephine and complete the work with Keen's rongeur, but I have here a set of much superior instruments,—namely, De Vilbiss's rotary bone-drill and rongeur. The drill makes twelve hundred revolutions per minute if required. You will observe I have made a circular ditch in the skull on each side, and the removal is completed under each isthmus of the scalp by the rongeur.

I have now removed a circular strip of bone nearly an inch in width without injuring either the dura mater or longitudinal sinus, and have left the island of bone attached to the scalp. The hemor-

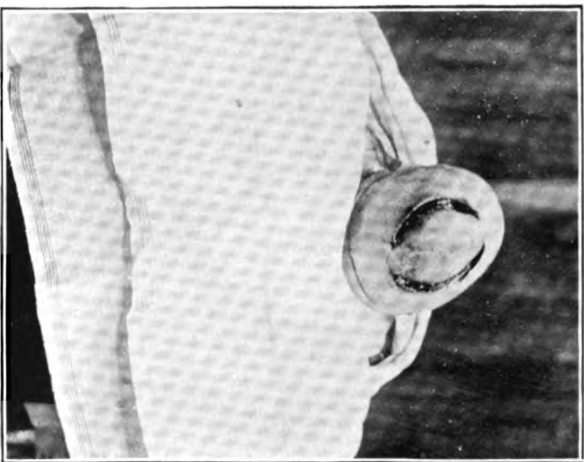


Fig. 7.—Cranlectomy for microcephaly.

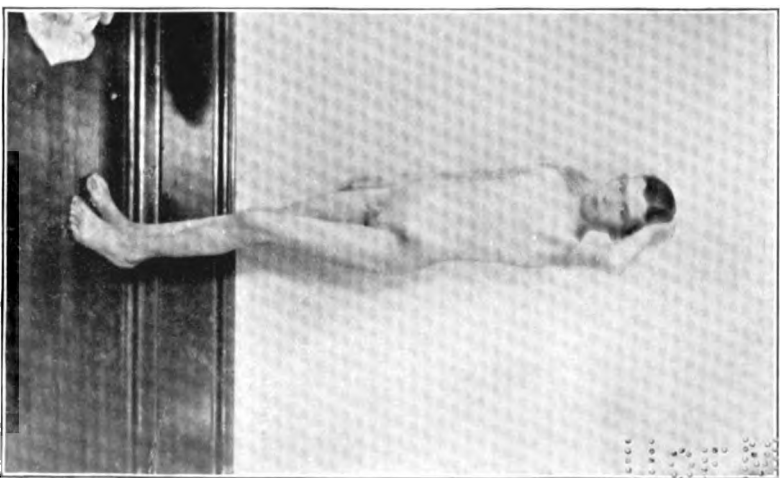


Fig. 8.—Tubercular abscess of the hip-joint.



३३३

rhage has been slight, owing to the efficient manner of controlling it, and before removing the rubber tubing I shall coapt the wound with silkworm-gut sutures and irrigate with a hot normal salt solution so as to prevent oozing. I shall, however, introduce a small strip of gauze on each side of the lower isthmus to provide for any oozing that may occur, and apply the gauze cap, which completes the operation. You will observe that the skull was fully a quarter of an inch in thickness. By this method of procedure we have given the greatest opportunity for brain expansion, and hope for some improvement in the mental condition of this child.

## TUBERCULAR ABSCESS OF THE HIP-JOINT.

CASE IV.—G. L., aged thirteen, has been under my observation for nearly a year. His mother brought him to me with pronounced symptoms of disease of the left hip-joint in the first stage. He was treated by fixation of the joint with a plaster-of-Paris splint extending from the ankle to above the umbilicus, with an elevated shoe upon the well foot, permitting him to walk about on crutches. This instrument after several months seemed to effect a permanent cure. About a month ago evidence pointed to a return of the difficulty, and about a week ago his mother brought him back with the appearance shown to you to-day (Fig. 8),—namely, knee flexion, slight inversion of the foot, prominence of the hip, limited and painful motion of the hip-joint, and a prominent bulging midway below Poupart's ligament, extending somewhat downward. Diagnosis, second stage with abscess of the acetabular variety, probably communicating with the pelvic cavity. Though, as a rule, abscesses below Poupart's ligament do not communicate with the pelvic cavity, yet authorities differ in this respect, and in this case it can be determined only by opening and exploration. As this abscess has undoubtedly ruptured with a marked tendency to burrowing, it would be proper to open, wash out, dress antiseptically, and be prepared to remove sequestra and make an excision if necessary. The incision is made obliquely downward and inward parallel with Poupart's ligament, between three and four inches in length, so as to permit a thorough evacuation and inspection. As I predicted, I find the abscess within the pelvic bones, but I am unable to find any sequestra, but enough erosion to justify thorough curettement of the perforations through the acetabulum. We will now irrigate copiously with a one to two thousand bichloride solution, following

it with a normal salt solution, thoroughly dry the cavity with gauze, mop out with an iodoform emulsion, pack and seal with strips of iodoform gauze,—first, however, putting in two or three silkworm-gut sutures, so as to lessen the size of the opening. The after-treatment will consist in removing from time to time the gauze and repacking as occasion requires. We have presumed that this case is tubercular, and will direct that he receives injections of nuclein daily, commencing with ten drops and gradually increasing it to fifty. My reasons for prescribing nuclein are that I have found it to be a valuable adjuvant to surgical interference in tubercular joint-troubles.

#### SUPPLEMENTARY REPORT.

Case I. made an uneventful recovery and the wire was removed on the thirty-sixth day following the operation.

Case II. suffered considerable shock for about forty-eight hours, and after this made gradual improvement without sepsis, leaving the hospital on the twenty-ninth day, the wound all healed with the exception of a small place which had to heal by granulation from the fact that the integument was insufficient to cover the wound at the time of operation.

Case III. left the hospital on the twenty-ninth day, the wound entirely healed, but with little apparent improvement in the mental condition. Improvement in the cases that have been reported has been slow and very gradual, so that if improvement does occur it will be a matter of the future. The child will be kept under observation and conditions from time to time noted. A later report states that the child died five and a half months following the operation from scarlet fever, and that there had been marked improvement in every particular.

Case IV. The result has been much better than was anticipated. When last heard from the wound had healed completely.

## EXTERNAL AND INTERNAL URETHROTOMY FOR RETENTION OF URINE; NEPHRORRHAPHY BY A FLAP METHOD FOR MOVABLE KIDNEY.

CLINICAL LECTURE DELIVERED AT THE SOUTH SIDE HOSPITAL.

BY J. D. THOMAS, M.D.,

Professor of Genito-Urinary Diseases in the Medical Department of the Western Pennsylvania University; Surgeon and Genito-Urinary Surgeon to South Side Hospital, Pittsburg, Pennsylvania.

---

GENTLEMEN,—The first case I present is one where retention of urine exists, due to strictures of the urethra; it is also complicated with fistulæ in the perineum.

I will read to you the history of this case as written up by Dr. Cavin, the resident, and I think you will find it quite interesting.

“R. G., aged fifty-six years; single; old soldier; admitted April 10, 1897. Patient has been since adolescence a slavish devotee at the shrines of Venus and Bacchus. His first trouble began thirty-one years ago by the formation of an abscess to the left of the anus. This was at first superficial and was lanced, but on its recurrence it was found to communicate with the rectum, forming a fistula-in-ano. Under palliative treatment this, the patient says, was cured.

“Patient acquired a gonorrhœa thirty years ago, which he allowed to go on without treatment. A long while after—he does not remember how long—he was seized with retention of urine, the retention occurring in the morning after sleep. This was relieved by an instrument. Following this retention he was treated by the passage of bougies for a time and experienced relief. Since discontinuing the treatment he has been subject to periodical attacks of retention, during which time his urethra was nearly or absolutely impassable to instruments and urine. After the attacks would pass off he would again be able to pass urine with some freedom.

“About one year ago he had an epididymitis.

“About fifteen months ago there appeared upon his perineum

three small ulcerating spots through which urine escaped during micturition. After this the urine escaped principally by the fistulæ. At times the fistulæ would close and then urination would become very painful, the urine dribbling away drop by drop, and the bladder becoming more and more distended. The pain on attempting to urinate was agonizing.

"It was in this condition, with the fistulæ closed and the bladder distended, that the patient entered the hospital, physicians on the outside being unable to relieve him.

"Several attempts were made, with small soft instruments, to catheterize him that resulted in failure. An attempt was then made with a small silver catheter, No. 8, French, which after about one-half hour of persistent effort was successful, and about forty ounces of urine were drawn from the bladder."

You notice in this history that there were numerous attacks of retention of urine that could not be relieved by instruments, but after awhile the patient could relieve himself by the natural method. We infer from this that when the patient indulged in venery or alcoholics there would supervene an inflammatory condition of the stricture or strictures causing a temporary swelling that for the time made the urethral canal impervious. After this temporary swelling subsided the patient could, in a manner, relieve himself. You observe that the doctor was able, after a prolonged effort, to get in a very small instrument. It is not always safe to make these persistent efforts for fear of damaging the urethra still more. In a case like this the best way to relieve the bladder temporarily is to aspirate. If this is done properly there is no danger.

The examination of the patient's urine shows that the kidneys are still in a healthy condition and the bladder not seriously impaired.

As the patient is now thoroughly anæsthetized, before operating we will proceed, after removing the dressings, to thoroughly examine his urethra.

The circumference of the penis is three and a half inches. This gives us, approximately, a urethra which will admit a No. 34 French. You notice the largest bulb admitted by the meatus is a No. 22 F., which indicates that the meatus is entirely too small. This bulb passes four inches, when it stops. Now, as I withdraw it I feel a number of irregularities, which convinces me that there are between

these two points a series of inflammatory exudates. No. 20 F. stops at the same point; No. 18 F. passes through this stricture, but stops at five and a half inches; No. 16 F. passes through this and stops at six and a half inches; No. 14 F. stops at the same point. I will now resort to soft bougies. You observe that I have to reduce these to No. 7 F., which I find passes all the way into the bladder. I will now remove this in order to pass a filiform, which will serve to guide a tunnelled instrument. My filiform will not pass; it has caught in some of the irregularities. I pass another alongside of it. This again catches. I pass the third filiform, which also catches. I will now manipulate these alternately, and you observe that I have succeeded in insinuating one into the bladder. After removing the other two filiforms I slip over the remaining one this tunnelled grooved sound until it reaches the deepest and tightest stricture. We will now place the patient in the lithotomy position. You observe about the middle of the perineum and to the right of the raphé two fistulous openings. On the left side, but away back near the anus, you notice a third opening. I now open the urethra upon the end of the sound, through the line of the raphé, and follow the filiform for some distance with the knife. The sound now passes into the bladder and I remove the filiform. I now pass a director along the sound into the bladder. The sound is removed when my finger is passed along the director into the bladder in order to thoroughly dilate the membranous and prostatic urethra. The Maisonneuve urethrotome is now introduced through the meatus and brought out through the wound in the perineum, and with its blade the entire floor of the urethra is cut. This makes room for the Otis instrument, which I now introduce and also bring out at the perineal opening. I open this up as far as it will go, without too much force, and find that it will only dilate to No. 30 F. I now draw the blade forward and cut the entire length of the roof of the urethra. This patient has a large organ, but he as yet has a urethral calibre adapted only for a small organ, so I again pass this Otis instrument, open it up to 34, and draw the knife forward. I now take this No. 34 F. steel sound in order to see if it will pass into the bladder. It is held too tightly at the meatus, so I take this bistoury and enlarge it somewhat, cutting below. You now see how readily the No. 34 F. sound passes into the bladder.

Most operators cut the anterior strictures before opening the

perineum. I prefer my own method, for it can be done quicker and with more precision.

Now we will turn our attention to the fistulæ. I pass this director through the upper one first, and it comes out deep in the wound. This I lay open freely. The second fistula, as you see, is connected with the one just cut. I now pass the director into the opening near the anus, and after passing it for some distance the point emerges at the urethral canal. The tissue here cuts like gristle and you can hear the knife grate. I now take the curette and scrape these fistulous tracks thoroughly. Although we have done a good deal of cutting, the hemorrhage is but moderate.

I now wish to draw your attention to the after-treatment of these cases. I do not introduce a soft catheter through the meatus and into the bladder and leave it there for a number of days; neither do I pass a drainage-tube through the perineal opening and pack around it. I simply leave everything alone as you see it now, but have the urethra irrigated several times a day with the Thiersch solution, and have the perineal wound kept thoroughly clean with a bichloride solution. The urine is aseptic, or soon becomes so, and this helps keep the parts sweet. If you introduce a catheter and pack the perineal wound your dressings are continually saturated with urine, which undergoes decomposition and makes the whole neighborhood of the wound foul. The catheter is uncomfortable, and will produce urethritis. The only occasion when I would resort to packing the wound would be when the bleeding demanded it.

A full-sized (No. 34 French) sound will be passed every third or fourth day until the urethra is healed. A bandage will be kept about the patient's knees to prevent too careless spreading of the thighs.

[NOTE.—Patient left the hospital on May 10, one day short of one month from date of operation. The recovery was perfect, with the fistulæ all healed. Patient could retain his urine for six hours, and stated before leaving that the ease and comfort he experienced when urinating seemed like a dream as compared with his years of suffering. A No. 34 French sound was passed on May 9.]

The next case I bring before you is one of movable kidney. The patient has a varied history, as you will notice when I read the notes taken down by Dr. Owens:

"Mrs. M. S., aged thirty-eight years. Some fifteen years ago patient gave birth to a six-months fœtus, which was followed by an

attack of puerperal fever. From that time has never been well. From 1882 until March, 1895, she had excessive floodings and pain.

"March, 1895, she was operated upon by Dr. Z., who removed the left ovary and tube. The ovary removed was found to be below and behind the uterus, the adhesions binding the uterus to some of the viscera. At this operation the doctor also stitched the uterus to the abdominal walls.

"The operation was followed by no relief whatever, and the same gentleman operated the second time, on April 17, 1895, for stitch abscess. One week after this, the patient says, pus in large quantities came from the vagina for several minutes. This was followed by a constant dribbling of pus from the same channel, lasting for a week.

"February, 1896, was operated upon by Dr. Y., who removed right ovary and tube, the operation being followed by complete relief so far as her pelvic region was concerned. But patient still complained of pain in left side and some pain at seat of cicatrix of first abdominal operation. Patient has always been 'nervous.' At location of pain on left side she sometimes noticed a 'swelling,' but paid no special attention to it, as she thought it was due to her ovarian troubles. Since removal of ovaries did not cause disappearance of her side trouble she again called upon Dr. Y., who made the diagnosis of movable kidney, which diagnosis was confirmed by other members of the staff. Patient requests an operation.

"At times patient passes small amounts of urine (sixteen ounces in twenty-four hours), at other times a normal quantity. Upon testing, urine is found to be normal in character."

Gentlemen, there is a difference between movable kidney and floating kidney: the former is acquired, while the latter is congenital. The former is not surrounded by peritoneum, while the latter is. The latter also has a mesentery, called mesonephron, while the former has not. Movable kidney occurs six times in the female to once in the male. This can only be accounted for by the tight lacing practised by females. It also occurs four times on the right side to once on the left, the liver being responsible for this.

Movable kidney is not at all uncommon. I am acquainted with a number of females, old and young, who have this abnormality. My attention has been drawn to some of these cases by the patients themselves, who happened to notice the "swelling" simply by accident, and not from any discomfort experienced. Others again suffer a good deal of discomfort and much pain. Where no pain is ex-



perienched these cases should be let alone. Where discomfort or pain is experienced something must be done. If a bandage will not succeed, then an operation must be considered. One of my patients suffers a great deal of pain, and at times passes large amounts of blood from the congestion produced in the kidney, due to twisting, probably, of the ureter. A bandage does not relieve her and she declines an operation. In cases of hemorrhage from the kidney it is well to remember this as a possible cause.

As you anticipate, the loose kidney may exist without causing any symptoms, and again they may be very marked and varied. The renal-plexus nerves are derived from the solar plexus. The disturbance of the renal plexus may reflect symptoms to all of the abdominal organs, so that in the case of a movable kidney we often find disturbance of the gastric and intestinal functions. We must not, then, lose sight of this possible cause. Pain may exist in the kidney itself or along the ureter.

If you find a movable body, kidney-shaped, in the abdominal cavity that can be pushed up into the place of the kidney you are at once led to the diagnosis of movable kidney, and if you can by exclusion eliminate every other condition that simulates this you are then positive of your diagnosis.

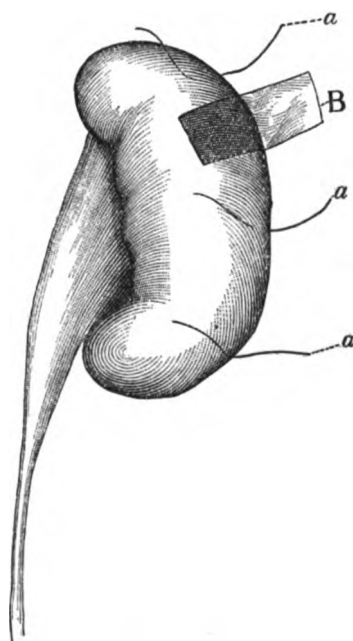
The patient is now ready for operation. You see how carefully the patient and everything connected with the operation is prepared. We do not intend to resort to drainage in this case, and aim to have healing take place under one or two dressings. The nurse places the pillow, tightly stuffed with sand, under the right side in order to elevate the ribs on the left side: by this means we get more room.

I place my hand on the back of the patient with my little finger on a line with the vertebral spines; now, if I make an incision along the index finger it will lead me to the anterior margin of the quadratus lumborum muscle, which will be my guide to the kidney. Another method for your line of incision, if it is to be a vertical one, is to press your fingers down on to the anterior margin of the erector spinæ muscle, and then advance them a little when you reach the margin of the quadratus lumborum. I can do that in this patient, but if she were very fat I would be unable to do so. Another method to reach the kidney is to make an incision below and parallel with the lower rib, beginning back over the erector spinæ muscle and going downward and forward to the iliac crest.

I now make a vertical incision from the ilium to the last rib,

going through skin and superficial fascia. The next incision carries me through the lumbar fascia. The few spurting arteries are quickly controlled by forceps and will not need ligating. I now cut through the middle lamella of the transversalis fascia, and I am able to show very beautifully the red fibres of this muscle. We will now pick up the inner lamella of this fascia and cut through it. I introduce my two index fingers into this opening and slide them, one up the other down, the entire length of the wound. With these retractors separating the wound I am able to show the kidney, for

FIG. 1.



Left kidney, anterior view. *a, a, a*, location of sutures. *B*, flap turned up.

in this case there is very little perirenal fat. I will incise this, and we are down to the fibrous capsule of the kidney. I now take hold of the kidney and move it up and down, and you are able to see what a wide excursion it takes. We were pretty safe in our diagnosis of movable kidney, because very few floating kidneys have ever been seen.

The kidney will now be held well up in the wound, and I will make two flaps from the fibrous capsule, one anterior, the other posterior, with the intention of bringing them up into the wound in the

back and securing them there, the object being to thus form two ligaments to serve as additional supports to the ligatures which I will introduce. I will first take a flap from the anterior surface, upper third, making it a little over one inch long and a little over one-half inch wide, the base reaching the mid-line of the organ. As there is a good deal of oozing, I will content myself with one flap in this case. This flap will be held in these forceps until I am ready to secure it in the wound. I will now pass three sutures underneath the fibrous capsule for the distance of three-fourths of an inch. You notice these ligatures, which are of kangaroo tendon, are first passed through some muscular tissue, then through the transversalis fasciæ, and before passing them under the fibrous capsule a "bite" will be taken in the fatty capsule (Fig. 1, a). I bring the needle out, passing it through the same tissues on the other side. Now, before drawing these ligatures tight the "flap" will be held up within the walls of the wound. A suture will now be passed through the flap, securing it buried in the wound. The wound will be finally closed with silkworm-gut sutures.

Various methods are adopted in doing this operation, which is called nephrorrhaphy or nephropexy. Some bury the suture some distance into the kidney tissue, others take up the fibrous capsule only in the ligature. Another is to split the capsule and turn it back in order to bring a raw surface in contact with the line of incision and transversalis fascia. Passing the upper ligature around the last rib is practised also. Other methods may be mentioned, but the very fact that so many devices are resorted to suggests that the operation, which is intended to *permanently* fix the organ, is sometimes a failure. This is true. In the present operation I expect to meet with success, for in addition to the ordinary proceeding I have insinuated between the lips of the wound a flap of tissue that will serve as a ligament, and this ligament will be continuous with the strong transversalis fascia. I would advise you never to pass your ligature deep into the kidney tissue. This tissue is friable, and your ligatures will cut their way up to the fibrous capsule before adhesions take place, and your kidney will soon be again movable.

This patient will be kept on her back for three weeks.

[NOTE.—This patient made an excellent recovery, the wound healing without infection. The period is too short since the operation to enable us to say anything with regard to the final result.]

## **RUPTURE OF A TUBULAR ANEURISM OF THE FEMORAL ARTERY.**

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY OF LOUISVILLE.

**BY W. O. ROBERTS, M.D.,**

Professor of Surgery and of Clinical Surgery in the University of Louisville; Surgeon to the Louisville City Hospital, etc., Louisville, Kentucky.

---

GENTLEMEN,—This patient has just been brought to the clinic by Drs. Pusey and Butler, who saw him for the first time yesterday afternoon. The history of the case as obtained from the doctors is about as follows: The man claims to be thirty-eight years of age; his occupation is that of a cook, and he has been connected with one of the large restaurants of the city for a number of years. He has been a steady drinker and has had syphilis. For some time he has complained of an uncomfortable sensation in the right thigh. Three weeks ago, while walking home, he slipped on the ice and, as he thought, simply sprained his leg. Pain was felt in the thigh. The following morning he suffered so much that he had to remain in bed, where he has been ever since until brought here. At first the swelling was slight and, as he says, confined to near the front part of the limb. His physician, a homœopath, treated him first for sprain, then for muscular rheumatism. The limb continuing to increase in size, the doctor told him he thought it must be a case of elephantiasis. For the last few days the swelling has increased very rapidly. When Drs. Pusey and Butler saw him they suspected diffuse aneurism and advised an immediate operation, and as the light in the patient's room was very poor, they thought best to bring him to the clinic.

You will notice that the right limb is very much swollen from the foot to the hip, the circumference of the thigh being about twice that of the opposite one. We can make out the outlines of a distinct swelling extending from about three inches above the patella to within two inches of Poupart's ligament, being located chiefly on the anterior, outer, and posterior surfaces. Distinct pulsation can be

felt along the inner border of the swelling, which corresponds to the course of the femoral artery. Upon auscultation we get simply a pulsation, without any distinct bruit.

Before bringing him to the clinic, Drs. Pusey and Butler introduced a small aspirating needle into the central portion of the swelling and drew off what was supposed to be bloody serum. The swelling is quite tense; there is no evidence of fluctuation, but there is some pitting on the anterior surface. The man's temperature is  $99\frac{1}{2}^{\circ}$  F.; his pulse is 112 and not of very good volume. He seems to be in a great deal of pain.

I take it that this is a diffuse aneurism, but am uncertain as to the vessel involved, and think it advisable to cut down upon it and endeavor to locate the opening in the vessel from which it sprang. The swelling extends so closely to Poupart's ligament that there are only two ways by which we can control hemorrhage with safety, one being to put a temporary ligature around the external iliac, the other to use the Wyeth pins and rubber tubing. We will try the latter. Dr. James S. Chenoweth will introduce the pins and manage the Esmarch; Dr. Butler will assist in the operation. While the patient is going under the influence of chloroform we will have the limb held at right angles to the body, so as to empty the blood-vessels as well as possible. Dr. Chenoweth having applied the tourniquet, I make a free incision along the course of the femoral artery. The tension is so great that you see as soon as I get through the skin there is a marked gaping of the lips of the wound. The parts beneath are deeply stained with blood. Upon carrying the incision a little farther upward, there is a gush of blood, which comes from the interior of the sac, which is formed by a fibrinous deposit. The upper portion of this sac has ruptured, and there is extravasation into the tissues. All of the soft clots, together with the fibrinous sac, I dig out with my hand, which leaves a space large enough to hold an ordinary cocoanut. After cleaning out the cavity thoroughly, I discover a small opening in the femoral artery at the upper extremity of Hunter's canal. This opening is near the lower extremity of a tubular aneurism of the femoral which extends to within one and one-half inches of Poupart's ligament. This tubular aneurism is somewhat larger than a man's thumb. I throw a ligature around the vessel immediately above and below the opening in it, and also one just above the enlargement in the artery,—that is, just a little below Poupart's ligament. Dr. Chenoweth will now slightly loosen the

Esmarch and we will see whether or not there is any hemorrhage. There is none, so we have undoubtedly found the cause of the trouble. As the man's condition is so bad, we will not prolong the operation by dissecting out the diseased portion of the artery, but will simply provide for thorough drainage and close the greater portion of the wound. In order to keep up the temperature of the limb, we will envelop it from the toes to the groin thickly in absorbent cotton held in place by a roller bandage, and direct that the limb be kept in a slightly elevated position.

While this man claims to be in his thirty-eighth year, his appearance would certainly indicate that he is at least ten years older. He has been, as I stated, a steady drinker, and has had syphilis. Idiopathic aneurisms usually occur in persons much older than this patient. Some people, however, age much more rapidly than others. Idiopathic aneurism is the result of an atheromatous degeneration of the coats of the artery, and among the chief causes of this trouble are age, whiskey-drinking, and syphilis. This aneurism of the femoral has no doubt been in existence for some little time, and at the point at which the opening occurred the inner coat of the artery had no doubt melted away rapidly, and the strain attending the fall was sufficient to cause a rupture; the opening being very small, an aneurismal sac of fibrin formed outside of the vessel, and when this ruptured the rapid increase in the swelling was noticed. In a short time this man would have had gangrene of the limb below from pressure of the increasing extravasation; inflammation would have occurred in the swelling; the skin would finally have given way, and the man would have bled to death. The best time to have operated would, of course, have been immediately after the swelling was first detected.

Very generally a condition such as this man presented would call for an amputation, because of obstruction to the circulation and pressure. I think, however, that in this case there is a good chance of the man escaping gangrene. The subsequent dangers attending such cases are shock, gangrene, sepsis, and secondary hemorrhage. Gangrene is due to the cutting off of the blood-supply to a part either by embolism, thrombosis, or the ligature; sepsis can be avoided by observing the strictest antiseptic precautions, and the same may also be said of secondary hemorrhage. Before the antiseptic era, secondary hemorrhage was a very frequent cause of death after ligation of arteries in the treatment of aneurisms because of the fact that the

ligatures had to slough through the vessels. Now, however, there is no sloughing as the ligatures become absorbed,—provided, of course, that strict asepsis is obtained. This also does away with the danger of sepsis, so that at the present day aneurisms, both traumatic and idiopathic, are treated almost exclusively by ligation. The methods most generally practised are those of Hunter, which consist in the ligation of the artery some distance above the seat of the tumor, or the complete excision of the tumor itself, the artery being ligated above and below the tumor. With the exception of traumatic aneurisms, my practice has been confined entirely to the methods of Hunter, and so far, while I have operated upon quite a number of cases, I have yet to meet with a failure or have any bad results follow the operation. This makes the fourth case that I have operated upon at this clinic, and, strange to say, three of them have been in the persons of negroes, only one in a white man. Two of them were aneurisms of the popliteal artery, one being popliteal and femoral, and the case before us. In two of them a ligature was put around the femoral artery in Scarpa's triangle; in the other I ligated the external iliac. In the latter case the aneurism extended well up into Scarpa's space from the popliteal. I first cut down just below Poupart's ligament, and, thinking the artery too much diseased, I went above and ligated the external iliac near the bifurcation of the common iliac. The case made an uninterrupted recovery. I saw the patient five years afterwards, and there had been no further trouble. In one of the other cases, a year after the operation I saw the man in the Bellevue Hospital, New York, in bed with an aneurism of the abdominal aorta. The other case I lost sight of after he was discharged. The case before us is the only one of diffuse idiopathic aneurism that has come under my observation. We will watch the progress of the case, and hope to present him at some future time showing a perfect result. Where it is practicable, it is best in these cases to confine the patient in bed with the limb elevated for several days before the operation is performed, and, of course, during and subsequent to the operation the strictest antiseptic precautions should be taken, and then we may expect, almost universally, good results.

[NOTE.—This man made a good recovery and was exhibited to the class a few weeks after the operation was performed. He is now manager of a hotel for negroes, and suffers no inconvenience from the limb. Pulsation of the posterior tibial can be felt at the ankle, but it is not so strong as that of the opposite side.]

## SYPHILITIC STRICTURE OF THE RECTUM.

CLINICAL LECTURE DELIVERED AT THE KENTUCKY SCHOOL OF MEDICINE HOSPITAL.

BY JOSEPH M. MATHEWS, M.D.,

Professor of Surgery and Clinical Lecturer on Diseases of the Rectum in the Kentucky School of Medicine; Rectologist to the Kentucky School of Medicine Hospital and the Louisville City Hospital, etc., Louisville, Kentucky.

---

GENTLEMEN,—The first case is one of stricture of the rectum in a woman. The question arises, What produced or caused this stricture? Is it of simple origin, or is there a deep-seated deposit? That there are benign strictures of the rectum there can be no question; that there are a few strictures that arise from simple causes cannot be doubted, but they are few. We may have a stricture of the rectum from traumatism by foreign bodies, pressure of the pregnant uterus, an ovarian tumor, or anything of that nature, but such cases occur very seldom; when you find a stricture of the rectum outside of these causes it may be regarded as a serious affection, the three factors being (1) syphilis, (2) tuberculosis, and (3) cancer. If you will reflect a moment, you will understand that any one of these would be quite a serious matter to your patient. We are going to show you this morning a woman who is, I believe, in good physical condition, yet she has a very serious stricture of the rectum. As the history is read to you, I desire that you will reflect upon it, and we will try and determine afterwards what the cause of this stricture is.

*History.*—The patient is a woman twenty-one years of age, and a prostitute. She gives a good family history. There is a history of syphilis of ten years' duration, for which she was treated by a physician for about two months at its incipency; since that time she has been treated on different occasions, but never for a great length of time on any occasion.

She applied at one of the colleges five years ago for the treatment of a growth,—she described it as like a cauliflower,—which was



situated in the anal region, and it was removed by an operation. She has noticed that the bowels have never acted freely since the operation, and in consequence she has been compelled to take some kind of medicine or else use enemas, sometimes both. Three months ago the bowels became unusually bound up, and she had considerable difficulty to procure an evacuation even by the free use of purgatives. Since then she has been compelled to strain violently with each evacuation, and the movement is accompanied by pain. She says also that a prolapse has made its appearance, which she notices after each movement.

*Present History.*—There is pain above the anus. This appears during an evacuation and continues for about fifteen minutes. She notices a protrusion with each action which will probably include the entire circumference of the anus. This she says feels very rough on its surface, being marked by hard substances underneath the mucous membrane. It always returns to the rectum without assistance. The bowels move only when stimulated by a cathartic or washed out by an enema. The actions are semisolid, broken into small fragments, and freely mixed with blood, pus, and mucus.

Patient had one miscarriage three years ago. Diagnosis, specific stricture of the rectum.

Now this is quite a complicated case. The woman, it appears, had performed upon her some time ago an operation for the removal of a cauliflower excrescence from the region of the anus. The very significance of the term, "cauliflower growth," indicates malignancy. The question arises, Was it cancer? There is also a history of syphilis. If we were able to ascertain the exact date that this deposition began in the rectum, we would have a better idea whether it was specific first and malignant afterwards, because it is a certainty that syphilitic ulceration of the rectum undergoes cancerous degeneration. I am sure that I have seen a number of such cases. If the trouble is malignant, it is more serious than if specific. I say more serious if malignant, inasmuch as the patient will die much sooner. As far as cure is concerned, the result would be about the same in either event, because whenever a stricture of the rectum exists for that length of time it becomes of a fibrous nature and is practically incurable. These growths appear to be nodulated; that is another significant term when you have a suspected case of cancer, because cancer is nodulated, sometimes indurated and nodulated. Then

she passes pus, mucus, and some blood from the rectum. It is true, she might do that from syphilitic ulceration. Notice another complication: she says at stool she has a protrusion, and she replaces it or it goes back of itself after the bowels have moved. What is this? Is it a portion of the cancerous mass, or is it a complication from hemorrhoids? I take it that the mass is hemorrhoidal. Now this raises an important question: if this woman has a malignant rectum accompanied by hemorrhoids, should you as a surgeon operate upon the hemorrhoidal condition? I say not, because if you make fresh wounds around the verge of the anus, it is very natural that it will take on more rapid cancerous degeneration. If a patient comes to you complaining of a protrusion from the rectum, even if there is considerable pain and inconvenience, if you find cancer of the rectum, I certainly would not advise you to operate upon the hemorrhoids. There may be a certain condition that you will meet where protrusion gives such great pain and distress that you are forced perhaps to remove the mass, but in the vast majority of cases where you find hemorrhoids complicating cancer, or *vice versâ*, it is best not to do any surgery on the lower part of the gut.

In the case before us, how are we to determine whether the trouble is cancer or whether it is syphilis? I will have you look at the patient, to see whether she has lost much flesh or whether she has a bad color; we will see if she complains of a great deal of pain, especially of a radiating nature, whether she has lost much blood, which are all characteristics of malignancy; on the other hand, we get a history of syphilis. Ulceration from syphilis of the rectum is rarely accompanied with much pain or hemorrhage; the deposition goes on gradually until it fills up the whole pouch of the rectum, forming a stricture. If there is a specific ulceration with stricture accompanied or complicated by hemorrhoids, should you operate for the hemorrhoids? Yes, there is no serious objection, simply because of the fact that it would make very little difference as far as the existing ulceration in the rectum is concerned. The points in this case are very close and difficult to make out. Is it a syphilitic affection, or a malignant affection accompanied with hemorrhoids? We will try to illustrate the distinctive features while the patient is under chloroform, and then determine what is the real nature of her affection.

If a cancer has existed for any length of time, there is, of course,

VOL. I. Ser. 8.—16

a constitutional infection, and therefore there must of necessity be a cachexia. There would be gland involvement, especially in the inguinal region. There would be great loss of flesh. In some cases of malignancy, however, no matter where the cancer is located, you will find that the patient will occasionally gain flesh. If they will change climate, eat freely, take tonics which improve the digestive functions, etc., you will find they take on flesh. This, however, is rare, and in the majority of instances they lose flesh, and do so rapidly. In this case there has been a moderate amount of hemorrhage. When ulceration takes place in malignancy you will usually have copious hemorrhages, a great deal of blood is lost. In this case the affection, we understand, began at the anal orifice; therefore we are led to suspect epithelioma, because there is where its seat usually is, as far as its incipency is concerned. Syphilitic ulceration of the rectum begins usually above the sphincter muscle in the mucous membrane itself by a deposition; therefore is a result of secondary or tertiary syphilis. It invades the structures slowly, insidiously, so much so that it may exist for two, three, or four years without bringing about any prominent symptom. But if there is an ulceration at the verge of the anus, which is operated upon by a surgeon as a "cauliflower excrescence," we might be reminded of another condition,—viz., chancroid.

A mooted question to-day in rectal surgery, and one that has been mooted for many years, is whether chancroids produce strictures of the rectum. I want to say to you that so great an authority as the late Samuel D. Gross used this expression in his excellent work on surgery: "Stricture of the rectum from venereal causes can only arise from an extension of chancroidal pus, and not as a secondary infection." Gosselin and his followers believed the same thing. Now, there is not an authority at present who does not believe that this is a mistake. Some few hold to the idea that chancroidal pus can cause a stricture of the rectum, but the vast majority of authorities, and even those I have mentioned, admit that it generally arises from a secondary or tertiary deposition. If this were at the verge of the anus, I would say perhaps it is a chancroidal ulceration, and perhaps underwent phagedenic degeneration, and consequently might produce a stricture low down if it were at the verge of the anus. If located above that point, I deny the proposition. We do not know what this patient's weight was a year ago; she is in comparatively good flesh now. Perhaps she has lost more than we would

naturally suppose from her present appearance. The best way to make an examination in a case of this kind is with the finger. It reveals by the sense of touch the character of the ulceration with which you have to deal; a speculum cannot do that. We see where the operation was done on the outside in taking off the growth which has already been described.

One observation that I would like to make while this girl is going under chloroform is: Sometimes you will examine a patient, man or woman, and detect a very close stricture, or I may say strictures, the whole lower portion of the rectum being blocked up and you can scarcely get your finger through, yet the patient may be in excellent physical condition. When this is true it cannot be malignant. To argue this question with you a little further, I want to ask you, if a patient has a deposition in the rectum to the extent of blocking up the lumen so that it will scarcely admit your finger, and looks well, is in good flesh, no cachexia, and has had the trouble five or six years, is it cancer?

I find that this woman has a distinct ulceration with an incipient stricture; the gut above is comparatively good and the sphincter muscle is not embraced. The muscle is so lax that I could push my whole hand into the rectum without any difficulty. Therefore we will content ourselves by believing that it is syphilitic, and will thoroughly curette the ulceration and break the stricture. Now, I want to call your attention to the fact that I do break these strictures in the rectum in opposition to the vast majority of authorities who have written on the subject, and I will argue the question with you after awhile and try to prove the correctness of my position. Pain about the rectum on manipulation is very severe and requires a great deal of chloroform. The stricture admits my two fingers, and I will make it admit my four fingers in a moment, then curette the ulceration thoroughly. I am going to break the stricture because the sphincter muscle is already partially destroyed. This stricture is within reach of my finger, measuring about three inches from the anus. You all know from your Anatomy that a stricture this distance from the anus does not embrace the peritoneum, and in breaking such a stricture we will not tear the gut, of course. I now have my dilator freely opened, so that I can run my four fingers into the gut, and I will show you that the patient will not have any serious hemorrhage. If this was malignant it would bleed profusely. Having now thoroughly dilated the stricture I will curette the ulceration.

We will insert a hard rubber tube into the rectum, surrounded or wound with iodoform gauze, which will allow all gas to pass freely, and be a guide also as to whether there is any hemorrhage above or not. The pressure of this tube will stop all hemorrhage that may take place. We will put over the anus a quantity of cotton and then a bandage, so the blood will escape if there is any hemorrhage.

In regard to plugging the rectum: You will readily appreciate the advantage of pressure. This is my own method of plugging the rectum: It is done by simply taking a hard rubber tube (not a soft rubber tube), wrapping it with gauze in the shape of a cone; opening the dilator, you push the plug into the rectum to the full extent of the gut, withdraw your instrument, and leave the plug *in situ*. The advantages of the tube are manifold: First, it is a valuable guide. Suppose we were to plug this woman's rectum, knowing that we have broken the stricture, perhaps torn a blood-vessel, how would we know whether the woman was bleeding or not? You may say we would get the symptoms, she would be nauseated, the pulse would be interfered with, she would show it in her countenance, etc. She might show these symptoms for hours from the effect of chloroform. Consequently you are anxious to know whether your patient is in this condition from hemorrhage (the blood cannot escape because you have plugged the rectum), or whether the symptoms are due to chloroform. By the use of this rubber tubing, the blood as it accumulates above will run through the tube and be your guide. Sometimes these patients lose from a quart to a gallon of blood.

Allingham's method is to take an aseptic sponge and pack it into the rectum through an instrument. You must understand that when you have hemorrhage in pathological tissue like this you cannot catch the artery and ligate it, partly because it is out of reach, partly because the structures are pathological and will not hold a ligature. It would be with the greatest difficulty that you would tie an artery in the rectum, and if you did it would tear through, consequently you must rely upon the plug. Allingham inserts a sponge, drawing it into an umbrella shape, and leaves it in the rectum, covering the sponge with powdered persulphate of iron. The objection to this method is that you have no guide, and you are therefore unable to tell whether your patient is bleeding or not; second, the sponge after a short time assumes the shape of a round ball and becomes very slippery; therefore pressure is not sufficient; it is not satisfactory;

it will roll around after a few hours; while by means of the tube, as I have shown you, it is held in place all the time.

In regard to the nature of the ulceration in the case before us: There is no indication of cancer, and a singular feature about the case is that she was operated upon for a growth just at the verge of the anus some time ago. What was the nature of that growth? If you will think for a moment I believe you will agree with me that inasmuch as this woman still has a protrusion, hemorrhoidal, and of the mucous membrane itself, the growth which was removed was probably of the same character. We notice a cicatrix here which looks as if it might have been produced by taking off a combined pile, partly external and partly internal; therefore it appears to us to negative the proposition of chancroidal ulceration or epithelioma, because there is no evidence of either one. This woman is constitutionally syphilitic; she has a secondary ulceration in her rectum; she is a clinic patient; but bear in mind that the best man or woman in the community may have the same thing. Consequently it behooves you to understand whether such conditions can be cured, and how they are to be cured or benefited. We believe this ulceration to be more or less in its incipient state. We mean that there is no special degeneration; there is not a stricture of the character that would be produced by deposition, involving two or three inches of the gut, but simply a stricture, which I have broken, involving the mucous membrane; there is not much deposition in the submucous tissue. The sphincter muscle is being undermined, as I say; it is exceedingly weak, if I may so express myself.

What are we to do in a constitutional way? This operation will not cure her. We broke the stricture and curetted the ulceration, but she must be put on constitutional medication. What shall it be? This deposition takes place as a secondary or tertiary manifestation of syphilis. I believe this is secondary, therefore the treatment for constitutional syphilis should be accorded this woman. You will say small doses of mercury. I am rather inclined to think that small doses of the bichloride of mercury answer better in these cases. We will give her one-fortieth to one-thirtieth of a grain of the bichloride of mercury three times a day and watch its effect, and we will also prescribe other specific medication, such as the iodide of potassium. It is foolish for you to undertake to cure a case of this kind unless you have the confidence of the patient, so much so that he or she will promise to remain under your observation for three years.

Patients often ask how long they shall take the medicine given them, how long they will have to remain under your observation. In cases of this kind the time should certainly not be less than three years, and they should not leave off the medication except by your direction. I would begin with ten grains of the iodide of potassium given in a glass of water (well diluted) three times a day, gradually increasing the dose say two or three grains a day until you reach fifty grains three times a day, making a total of one hundred and fifty grains to be taken during the twenty-four hours. As I told you in a previous lecture on this subject, you will meet some people who have an idiosyncrasy and who cannot take iodide of potassium in such doses. The question arises here, will a man who has an idiosyncrasy and can be gotten under the effect of iodide of potassium by giving it in three-grain doses derive the same benefit as a man who has not the idiosyncrasy and takes the routine treatment for syphilis,—in other words, where you can increase the dose to one hundred and fifty grains a day? I am candid in saying that I do not know; but I say, more's the pity if he cannot take it, for look at the ravages that this woman will suffer if she is unable to be medicated in this manner. The sphincter muscle will be destroyed; she will have not only one, but a half-dozen strictures, which will block the rectum; she will be unable to void her feces; she will finally die as a result of stricture of the rectum. Therefore watch your private patients, see that they increase the dose gradually and steadily, and watch its effect upon them.

What shall you do in the way of local medication? It should be just the same as if you had an indolent ulcer elsewhere, because this character of an ulcer is indolent; it does not show a disposition to heal. What would you do if you had a syphilitic ulcer of the leg? You would endeavor to inflame it; in other words, you would establish in that indolent ulcer a disposition to heal by some procedure that would cause granulation. We scraped this ulceration, or curetted it, partly for that reason. But in your local applications after this you do not want to administer chloroform, therefore by gentle divulsion you can make your local application directly to the ulceration. What shall it be? The favorite remedy for touching up a condition which simulates this surface would be iodine; but you cannot use iodine here, it would cause such intense pain; however, you can use carbolic acid, and you can use it pure, because, being inside the true skin, you can make your application, and while

there will be a stinging pain for a few minutes, it will last a very short length of time, and it is one of the best of applications. You can use a solution of the nitrate of silver, say forty grains to an ounce of water, or you can take a stick of lunar caustic and touch up these ulcers, or this surface of ulceration, just as you would anywhere else,—in the throat, or on the surface of the body, tongue, mucous patches, etc. Anything that will stimulate an indolent ulcer elsewhere will be a good application for such a condition within the rectum. But what you want to rely upon most is internal medication to get this deposit reabsorbed by nature. I have no doubt that in the early stages of syphilis this can be accomplished, but if you do not see your patient until the fibrous condition is established involving the whole lumen of the gut the effect of medication is *nil*. Allingham says in his most excellent work that a patient with cancer is more fortunate than this, because he will die earlier. The tortures that come after the rectum is entirely blocked you can quite well imagine: then begin fistulous tracts; feces come down upon the stricture, and as a result we have a fistulous communication on the outside. I recently presented before this class a case of that kind resulting from cancer of the rectum, and you may have the same thing from syphilis. These fistulous tracts not only run down to the nates, but go often into the perineum, back to the sacrum, down to the thigh,—indeed, they may run in any direction. If you were to operate upon such a case for fistula, you would certainly make a mistake if you did so without first examining the inside of the rectum. When you have stricture from any cause to deal with, do not operate upon the fistula until you have made a thorough examination and looked after the stricture. Relieve the stricture from above by either gradual or forcible dilatation. Which is the best? I ask the question in all honesty, because I have written often about it and have been opposed by some very eminent men. My friend, Dr. Bauer, of St. Louis, an old and distinguished surgeon, says that if he were to treat a stricture in the manner I have described and practised he would injure the gut,—in other words, that he would tear into the peritoneum. How could he do that? Sixty out of sixty-five rectal strictures are within reach of the finger. There is no peritoneum at that point. You know that the lower half of the rectum does not float, it is attached; therefore how could he tear into the gut? It is impossible. I say, then, whenever you have a stricture that you can engage with the finger, that it is below the



peritoneum, that it does not involve the peritoneum, that it is much better to forcibly dilate it than to practise gradual dilatation. Why? You can take a stricture of that character, and gradual dilatation requires that the patient should report to you at least three times a week, and the introduction of a bougie for dilatation is exceedingly painful. You insert it to-day; the patient comes back in three days and you find no change, it is about the same. You may insert it three times a week for three months, and you will scarcely detect any change; there may be a little. After six months, if the patient has escaped you during that time for one month, there is a recontraction, and it is just as bad as when you first began. If he has not escaped you, you will not notice any material change, especially where there is a succession of strictures in a syphilitic patient. Therefore if you can accomplish in three minutes by forcible dilatation what it would take many months to accomplish by the method of gradual dilatation, why not do it? Let me illustrate: Suppose you have a stricture that embraces three inches of the gut, and you have an opening through it half an inch in diameter, which will just admit the tip of your finger,—a fibrous condition; then you insert this dilator through the opening, using only such force as may be necessary, and dilate it instantly. I have never had any trouble following such dilatation. Neither will you. Therefore I see no objection to dilating to that extent in one minute, obtaining a result which it would require six months to accomplish by the method of gradual dilatation.

## ACUTE OSTEOMYELITIS IN YOUNG CHILDREN.

TWO CLINICAL LECTURES DELIVERED AT TUFT'S COLLEGE MEDICAL SCHOOL.

BY CHARLES GREENE CUMSTON, B.M.S., M.D.,

Assistant Professor of Surgical Pathology, Faculty of Medicine, Tuft's College; Fellow of the American Association of Obstetricians and Gynecologists;

Corresponding Member of the Association of Genito-

Urinary Surgeons of France, of the Patho-

logical Society of Brussels, of the

Electro-Therapeutical So-

ciety of France, etc.

---

### LECTURE II.

GENTLEMEN,—In our last lecture I dealt with the etiology and pathology of osteomyelitis in young children, and to-day we will consider the symptoms and treatment of this affection.

The symptoms may be divided into general and local. The commencement of the disease is rarely seen by the surgeon, especially in the newly born, as the beginning is slow and insidious and the crying and restlessness of the infant are not usually heeded, although sometimes the parents remark that their baby no longer sleeps, and that it nurses infrequently and only a little at a time. Sometimes fever is present at night, and diarrhoea or constipation are met with equal frequency. Vomiting is rarely noticed, but the principal symptom complained of is the pain which the slightest movement will provoke. In very acute cases with a sudden commencement, in which the fever is very intense or where serious symptoms appear at once, convulsions are often observed in the newly born, while delirium is present in older children.

When you first see these patients they are not usually emaciated, but are of normal weight and present no hereditary tendencies. When the latter do exist it is sometimes syphilis and at others scrofula, and it is especially in the latter class that a cachectic con-

dition is met with. The lips are red, dry, and cracked; a considerable elevation of temperature is often found, and always an extreme frequency in the pulse-beat; but it may happen that none of these symptoms are present, and that pain is the first symptom observed.

Now, how shall we find out the presence of this pain? In the first place, you all know that the presence of the physician is sufficient to cause children to cry, and as crying out is the only indication which we have to guide us as to the point at which the lesion exists, it is of the utmost importance to prevent them from crying excepting when pain produced by palpation causes them to do so. Consequently the first thing to do is to quiet them, and when this result is obtained you may commence your examination. Now, if the latter is painful, the child will immediately commence to cry at the slightest touch, and then you must try to win its confidence. In order to do this, you should never begin by the examination of the diseased limb, which, as I shall point out to you, can be distinguished at first sight.

You should begin to palpate the healthy limb, always beginning at the foot and slowly going up towards the thigh. This manœuvre should be repeated several times if the child cries, and as no pain is produced he will soon desist, when the diseased limb may be examined. If the patient is lying on the bed, as is usually the custom, you should depress the mattress with the hand until you can push it under the child, after which the limb should be slightly raised with great gentleness. The child, being now used to you, will only cry out when you cause it pain, and the cries will increase in intensity as the pain becomes more acute, thus indicating the seat of the lesion. Now, in order that you may be sure of your diagnosis, you must proceed rapidly, because after a short time the child will cry continually, and it then becomes impossible to distinguish what point is the seat of the greatest pain.

I said that the diseased limb was easily distinguished, and two symptoms will be present to aid you,—namely, immobility and the peculiar position of the segment of the limb which is the seat of disease. The child will not execute the slightest movement with it, and often will keep it quiet even when it is pricked, as if it were paralyzed.

If the little patient is raised up, the limb will hang down as if paralyzed. When the child is lying down, he remains in a particular

position, which is always the same for the segment attacked: semi-flexion and outward rotation for the thigh and leg; slight abduction for the arm; semiflexion and semipronation for the forearm.

Swelling appears almost at the same time as pain without any known cause for its production. The parents will tell you that in the morning when they undressed the child they found it present, when the night before there was nothing. It is, in the first place, circumscribed in the neighborhood of one of the extremities of the diaphysis, and then, little by little, it extends in surface. It increases both in length and in breadth, and soon completely surrounds the limb, involving the nearest joints. It may even happen that in cases in which there is a commencing arthritis the swelling appears first around the joint, and only spreads over the limb later on.

For a certain time the skin preserves its normal color, and later it becomes a milky white, in which may be seen the dilated veins. In other cases it may become shiny and red and present all the characters of a lymphangitis. This is especially met with in osteomyelitis produced by the streptococcus, and you will easily understand the reason for this because I have already pointed out to you that this micro-organism has a predilection for the lymphatic system. By palpation you will find a hard and resisting œdema in the beginning, but it will become more and more soft until fluctuation is present.

If you are dealing with a case of osteomyelitis seated in a deep bone, such as, for example, the femur, the greater number of these symptoms will be wanting. Tumefaction does not exist, and the shape of the region will not have changed; the skin has its normal color, and it is with difficulty that you can make out tension or resistance in the soft parts. But as the disease continues its evolution the symptoms become accentuated, and after a short time they will be easily made out.

We now come to the second period of osteomyelitis, and let me say at once that this division into two stages is simply made to render the subject more easy of comprehension, because the clinical signs of this disease rapidly succeed one another without any interval between them. The swelling continues to increase, giving a peculiar shape and considerable size to the limb. The skin still keeps the color that it had in the beginning, but becomes infiltrated and œdematous, and the dilatation of the veins becomes more marked; im-

mobility is still more complete and the position of the limb more characteristic.

During this time the suppuration in the bone has progressed, and you may now be able to get fluctuation. But you may not always be able to get this fluctuation, even when pus is present. In these cases there is another sign that is always well to look for; this is the periosteal lump, the mode of formation of which I have already indicated. According to Lannelongue, this is a favorable sign, because it indicates the formation of a barrier which for some time opposes the invasion of suppuration, and which allows us to affirm the presence or absence of a subperiosteal collection.

If a marked depression is seen beside the swelling, you may be sure that there is a detachment of the periosteum and a subperiosteal abscess. If, on the contrary, the surrounding tissues insensibly blend into the swelling, you may be nearly sure that no subperiosteal collection is present. It may also give us another hint when it is present, because it is then easy to surmise whether the subperiosteal abscess completely surrounds the bone.

When the periosteum breaks and allows the suppuration to progress, the latter tends to come nearer and nearer the surface of the limb, and then you will witness all the phenomena of phlegmon, the symptoms of which I will pass by, believing that you are all sufficiently familiar with them.

These abscesses open exteriorly when they are not incised, and a variable quantity of pus is evacuated. Sometimes it is frankly phlegmonous without any particular odor, sometimes it is fetid. Chassaignac thought that this odor was due to pieces of necrosed bone-marrow which the pus held in suspension. Very infrequently the collection may not be made up of pus, and a case is reported in which only a large quantity of liquid blood was found. A case is reported by Gussenbauer in which the exudation consisted of a very hemorrhagic serum which, although not purulent, contained micrococci.

The complications of osteomyelitis are characterized by symptoms which are easily recognized. Detachment of the epiphysis is accompanied by a complete impotency of the limb, and if you move the diseased segment you can produce movements which do not normally exist. You might mistake them for a fracture or a dislocation, but I will point out to you the means for avoiding this error.

Arthritis is also easily made out, especially when it occurs after the beginning of an osteomyelitis. There is œdema around the joint, and pain is produced by the slightest movement, and the diagnosis is rather difficult only when this complication takes place at the beginning of the disease. At the elbow and at the hip you should look for the symptoms of detachment of the epiphysis because there is a cartilage of connection in the synovial cavity. In other joints it is only by a careful examination that you will be able to make out the cause of the arthritis. You will be informed when by palpation you find a hyperostosis; the same for fracture of the diaphysis, the symptoms of which are those of all complete fractures. A sequestrum causes a continued suppuration, and the formation of a fistula and the simple passage of a probe will tell you everything.

The flat bones in young children present certain peculiarities that appear to me interesting to mention. Osteomyelitis of these bones appears more frequently in young children than in adolescents. The frontal bone, sacrum, pubis, ribs, and ilium may each be the seat of the disease, and the symptoms are the same as those of the long bones; but in the sacrum, for example, an abscess may develop in the pelvis instead of at the exterior, in the lumbar region.

Osteomyelitis of the ribs is very frequent in adolescents. In little children only one abscess usually exists, while in adolescents you will generally meet with two,—one anterior and one posterior. They may lead you to believe that there is a pleurisy because they give dulness on percussion and ægophony, as in pleural collections, but knowing this you will easily avoid making a mistake.

Of all the bones the pubis is the most infrequently the seat of disease in older children, but it is as frequently attacked as the sacrum and ribs in young subjects.

When an osteomyelitis is seated on the iliac bone it usually produces an external swelling. For the moment I will say nothing of osteomyelitis of the frontal bone, because I shall speak of this at the end of the lecture, when I consider the question of the relationship between this disease and syphilis.

The diagnosis of osteomyelitis is always a difficult matter and even impossible at the beginning in children and the newly born, although you may examine most attentively. The initial symptoms resemble those of many other diseases, such as typhoid fever, meningitis, rheumatism, deep phlegmon of the limbs, and coxalgia.

When you see the patient at the beginning of the general symptoms, your first thought will be of an infectious disease. The character of the pulse, the appearance of the tongue, the lips and expression, and the diarrhœa all lead you to suppose that you are in the presence of typhoid fever, because you know that this disease is far from being infrequent in young subjects, and the error can only be avoided by making a complete examination of your patient. The convulsions and delirium may make you consider the question of a meningitis, but as other symptoms of this disease will be wanting you can eliminate it.

When the commencement of the disease is less marked and the general symptoms pass by unnoticed, and when palpation has demonstrated the presence of a painful point at some part of the body, your diagnosis will be less liable to be at fault, but, nevertheless, it is not always easy to make. The cries of the child locate the seat of the pain near the joints, but this may lead you to believe that the case is one of rheumatism.

When you see the swelling appear a little farther beyond the joint, the diagnosis of rheumatism may be eliminated. When you will have found that movement is not painful and, still more, that the fever is higher than in rheumatism, your diagnosis will be still more certain. But this will not be the case when an arthritis is the first apparent symptom, and this is nearly always the case in the elbow and hip. In these cases you should look for the signs of detachments of the epiphysis, while in all others you will recollect that acute arthritis is very rare in children under two years, and by carefully searching you will always be able to find the cause of the joint-affection.

To differentiate osteomyelitis from coxalgia is nearly impossible in the beginning, especially in cases of subacute osteomyelitis in which fever is wanting. The history of the case will be of little use and limping is almost never observed, consequently before pronouncing the nature of the trouble you must wait until the disease has gone through its evolution and carefully watch for the appearance of local and general symptoms.

As to deep phlegmon of the limbs, it is to be distinguished from osteomyelitis by the redness and heat of the skin, as well as by an early fluctuation which accompanies the latter affection. A mistake, it is true, will present no great inconvenience, because early incision

is the treatment for both these diseases and has the advantage that it will show whether the medullary canal is invaded, and it always is if after this opening the local phenomena do not get better and the pain still continues.

Now the question comes, are we able to know before any microscopical examination has been made what microbe has been the cause of an osteomyelitis? Lannelongue has given some distinctive characters, and in a large number of cases he has been able to predict the organism which bacteriological examination demonstrated later. Osteomyelitis produced by the staphylococcus has a classical evolution, with which you are all familiar and which serves as a type for the description of the disease. It is nearly always accompanied by a dilatation of the superficial veins, but this is not a pathognomonic sign, in the child at least, because a case has been reported in which an osteomyelitis caused by the pneumococcus presented this condition of affairs.

Osteomyelitis produced by the streptococcus has a more rapid beginning, and the intensity of its symptoms recall the most serious forms of the affection. The fever is very high and has greater oscillations in the morning and evening than when the disease is due to the staphylococcus. It also falls more rapidly, and at the end of three or four days, when the pus has collected, it will be found to have completely disappeared.

The lesions that it produces are less marked and less extensive, while deep phlegmon rarely is present, and the pain is not so intense. Now, as we know that the streptococcus likes the lymphatics, instead of the venous engorgement that is produced by the staphylococcus, you will see a characteristic red color appear very early, due to an angioleucitis or an erysipelas which is nearly always accompanied by an adenitis. The pus is serous, less heavy, and not so greenish in color as when produced by the staphylococcus. Arthritis is more frequent, but detachment of the epiphysis, the formation of sequestri, and visceral complications are also not so frequent.

Courmont and Jaboulay, who have experimentally endeavored to differentiate between a staphylococcic and streptococcic osteomyelitis, have come to the following results:



**STAPHYLOCOCCIC OSTEOMYELITIS.**

Animals begin to be sick two days after inoculation.

Rectal temperature varies between 40° and 41° C.

Painful tumefaction in one or both knees.

Death in eight days.

*Autopsy.*

Periostitis.

Large sequestri.

Slight suppuration of the juxta-epiphysary region.

Purulent arthritis frequent.

Integrity of the cartilages of conjugation and of the marrow.

Abscess of the kidney always present.

Abscess in the muscles, especially of the heart, frequent.

**STREPTOCOCCIC OSTEOMYELITIS.**

Animals are sick the day following inoculation.

Rectal temperature is above 40° C.

No tumefaction of the bones or joints.

Death in six days.

*Autopsy.*

No periostitis.

No sequestri.

Profuse suppuration of the juxta-epiphysary region.

Purulent arthritis very infrequent.

Bone-marrow always invaded.

Abscess of the kidney frequent.

Muscular abscess absent.

The pus and blood from the heart gave pure cultures of the streptococcus.

From these results they conclude that the staphylococcus directly attacks the bone-tissue of the juxta-epiphysary regions, producing necrosis, periostitis, and a very slight suppuration, sometimes an arthritis.

Secondly, that the streptococcus directly attacks the bone-marrow in the juxta-epiphysary regions, leaving the bone-tissue, periosteum, and the joints intact.

Koplik and Van Arsdale have obtained nearly the same results, excepting in regard to the arthritis, and they say that in certain cases of acute articular suppuration, more often polyarticular, occurring after wounds it is certain that the surgeon has to deal with a hidden osteomyelitis due to the streptococcus when the joints are involved secondarily, and that there is hardly any clinical symptom at the primary focus. This is probably an osteomyelitis due to the streptococcus.

Dardenne mentions three cases which corroborate in certain respects the experiments of Courmont and Jaboulay. Injections made with cultures of the pus taken from one case produced a suppurating arthritis of the knee in the rabbit; in another case he mentions the

presence of a sequestrum and a fistula, while in a third he met with suppuration in several joints.

In the presence of such facts we may perhaps be permitted to believe that we are not able to distinguish in every case at first sight these two types of osteomyelitis. The venous engorgement is not peculiar to the staphylococcus any more than are the arthrites, consequently as yet we cannot base our diagnosis on the evolution of the disease, on the intensity and progress of the fever, the gravity of the general symptoms, and the existence of an angeioleucitis for the streptococcus.

Pneumococcic osteomyelitis is less frequent and its clinical progress is less well established. Nevertheless, the cases that have been reported show that the beginning is very marked and that the general condition is serious. It is accompanied by a sharp reaction, considerable swelling, and detachment of the epiphysis and arthritis. As I have already said, one case presented a very dilated superficial venous net-work, but the presence of a subperiosteal abscess was not found. But what it offers in particular is that in spite of the rapidity of its lesions they disappear very quickly and recovery takes place in a short time. At the autopsy of a case a fibrous ankylosis of the hip-joint was found. The bone lesions produced by this organism are similar to those that it produces in the other organs; they are rapid and intense, but repair quickly because the virulence of this microbe is very quickly attenuated.

Osteomyelitis due to Eberth's bacillus is very rare in young children, and when it does occur it has a subacute evolution. The beginning is so very insidious that it often passes by unnoticed. Febrile reaction is very moderate and may even be completely wanting. The lesions in the bone are always limited, and in some cases the abscess produced is filled with a serous liquid. Necrosis is infrequently observed and is always superficial and never forms a true sequestrum.

The local complications of osteomyelitis are often difficult to diagnosticate. As I have said, detachment of the epiphysis may be mistaken for a fracture or a dislocation, but you will have the general symptoms which always exist in an osteomyelitis and which are not present in cases of fracture. You should also remember that the latter are rarely complete in young children, and even if you should believe that you have an exceptional case, you will not find crepita-

tion in detachment of the epiphysis that is so characteristic as that of a fracture. The pus with which the extremities are surrounded prevents this sign from being present.

As to mistaking a detachment for a dislocation, you should always remember with what extreme infrequency the latter occurs in children. If you exercise traction on the bones of a newly born child, you will always produce a detachment of the epiphysis and never a dislocation excepting at the shoulder, where the latter has sometimes been observed to occur.

When you are in the presence of a suppurating arthritis you should always search for the cause in the bones which form the joint, because acute spontaneous arthritis does not exist in young children.

The visceral complications, such as nephritis, pericarditis, and bronchopneumonia, are accompanied by the same symptoms as in all patients, and I would only remind you that dulness on percussion and ægophony are not always sure signs of a pleurisy, because an osteomyelitis of the ribs may also give rise to these signs.

I have already told you that the lesions of osteomyelitis have sometimes been taken for those of hereditary syphilis, and, in fact, this mistake is quite possible in young children. You all know that the infantile pseudoparalysis described by Parrot closely resembles the immobility of the limb which occurs in the beginning of osteomyelitis, and what is more, syphilis, like osteomyelitis, attacks several bones at once in young children. Now, can we differentiate the two diseases?

You must always carefully examine the hereditary antecedents, and if these give you no clue you must examine the child and endeavor to discover if there is no sign of syphilis present, such as mucous patches, eruptions on the tongue, coryza, or scabs around the nostrils. These signs hardly ever are wanting, but usually the replies of the mother made to your questions or the examination of the child will enable you to be sure as to the existence or the non-existence of syphilis.

Supposing you have a child who is manifestly syphilitic and who presents lesions of the bone. Your most natural tendency would be to consider them due to the latter disease, but you must know that osteomyelitis and syphilis often exist together in the same subject.

The former malady is usually primary; that is to say, it is seated in a bone in which syphilitic manifestations have not as yet occurred,

consequently you must always be careful not to make a diagnosis of syphilitic pseudoparalysis by the simple symptom of immobility. Without doubt there is a solution of continuity in both cases, but in each one of them the progress of the disease and the phenomena which accompany each one of them are very different.

I have pointed out to you that there is nearly always a prodromal period in osteomyelitis. The child continually cries, does not eat, and often has diarrhoea. All these symptoms are wanting in syphilitic pseudoparalysis. Then when immobility has been found the condition of the little patient remains the same, its limb has the same aspect, and its general condition does not change, while in osteomyelitis a swelling soon occurs which takes on enormous dimensions.

But the most important symptom of all is the presence of fever, and in the majority of cases it alone will be enough to allow you to make a diagnosis. In osteomyelitis it is always present, even in the beginning, while in syphilis it is almost always wanting.

The coexistence of an osteomyelitis and syphilis can easily be admitted, for not only does the latter disease predispose young children by weakening them, but it provides entrances for microbes which later on invade the entire organism.

The case reported by Koplik is that of a child two weeks old whose mother had had repeated miscarriages and was treated by biniodide of mercury. The symptoms of osteomyelitis were quite marked during life; the temperature was high; the right knee swollen and painful on palpation, and gave vent to a half-ounce of pus when incised; the left hip contained a purulent collection, and there was necrosis at the ends of the bones with detachments of the epiphysis. Microscopical examination showed the streptococcus, which had probably entered the organism through the wound of the umbilicus, which was red and inflamed some days after the birth and which later on suppurated. An exploratory puncture practised in the left swollen knee gave issue to some serum which did not contain any organism, which is quite an astonishing fact.

If you will consider that this tumefaction existed on the side of the knee in a syphilitic child, you might be induced to believe that it was a gumma of the bone which later suppurated because it had been invaded by the streptococcus. But if it had been thus, it would have been a second manner of the occurrence of osteomyelitis in a

syphilitic child; it would have been an osteomyelitis following an infection of a gumma and developing in the bone already the seat of syphilis.

Several cases have been reported in which the frontal bone was the seat of disease, and you all know that syphilis has a predilection for the bones of the skull in children, and it has been demonstrated that osteomyelitis can also develop there. The prognosis in these cases is always extremely serious on account of the meningeal lesions that may arise, and consequently energetic treatment is indicated without delay. Here especially free resections are indicated, but you should never neglect specific treatment, because it may prevent an end which is usually fatal.

We now come to the consideration of the treatment. In 1876 Ollier called attention to trepanation of the bone, which had long ago been practised by J. L. Petit and Van Sweiten, as well as to the great number of its indications, and considered it as a means of aborting osteomyelitis. He also said that it would cut short the infectious symptoms and prevent necrosis from increasing. Lannelongue goes still further, and says that trepanation is the only method whose usefulness and indications are positive.

Kirmisson also advises early exploratory trepanation performed with a small trephine. If pus flows out, trepanation should be performed, but if no liquid flows away this little operation will act like a bleeding of the bone, which is most excellent.

To-day it may be said that two operations are still in vogue. The first was advocated by Berger, Poncet, and Saint Germain, and is as follows: when you have found fluctuation in a case of osteomyelitis you should incise the subperiosteal abscess, irrigate, and drain. If the general symptoms do not improve after this first operation, you may then trephine the bone.

Lannelongue's method is simply the application of the trephine. The affection always having its origin in one of the extremities of the diaphysis it is there that we should first trephine, but a simple opening is usually insufficient, especially if a detachment of the periosteum and periosteal abscess extend over a certain length of the diaphysis, thereby necessitating a second application of the trephine.

Now, in osteomyelitis as in any other surgical disease, one method is not sufficient, because each case has its own particular indications, and I will indicate the proper treatment for each one.

If you are called to see the patient at the beginning of an osteomyelitis, there is as yet no pus in the diaphysis, and consequently an immediate application of the trephine will render the greatest service. When the button of bone is removed, you will see one or two drops of purulent liquid come out from the interior of the bulb, and with proper drainage you will nearly always bring about a cure.

Now, if you are called later, after a periosteal abscess has formed, trephining over the bulb is still indicated, but in cases in which the detachment of the periosteum and periosteal abscess covers a large extent of the diaphysis two or three applications of the trephine will be necessary. We know that in young children the medullary canal does not extend quite up to the bulb, and the suppurating central marrow cannot run out by the first opening made, consequently the general symptoms do not get better, and the same dangers exist after the operation as before.

It is not the same when along with the periosteal abscess you find a detachment of the epiphysis. In this case a simple incision is sufficient providing there is no inflammation in the medullary canal. The pus contained in the bulb and in the epiphysis can easily burrow outward, and it is especially in these cases that the application of the trephine is to be employed, and the only cases in which it gives good results.

But your interference should not stop here. You must see that there is a good coaptation of the separated fragments, a very good apparatus being made by small pasteboard splints well padded with cotton, between which the ends of the segments of the limbs to be immobilized should be placed in order that callus may form in good position. It is only later, when suppuration is about to finish, that a plaster apparatus is to be applied.

When an osteomyelitis is complicated by arthritis, besides the trephining the joint should be freely incised and drained, and you should not hesitate to make several openings if the flow of pus is not perfectly free.

You know that there are three forms of arthritis; first, arthritis by terebration of the diaphyso-epiphysary cartilage; secondly, the arthritis produced by a separation of the epiphysis; and, thirdly, arthritis by propagation of the inflammation from the subperiosteal layer or intermuscular tissues, and it is only in the first variety that resection should not be resorted to.

When suppuration has invaded the medullary canal, whether there is or is not periosteal abscess, separation of the epiphysis or arthritis, you may apply Tscherning's method, which consists in a curettement of the bone-marrow. After opening the medullary canal all the marrow that it contains or that is supposed to be the seat of suppuration is removed by the sharp spoon. In every case drainage is necessary.

The only proper treatment of osteomyelitis of the bones of the skull is total resection, because there is always pus on the under surface, which may cause fatal cerebral complications.

As to resection of the long bones when necrosis or sequestra are present, it is rarely indicated in young children, as the necrosis is usually superficial and of small extent.

Ollier advises expectation in the majority of cases because it is better to allow the sequestrum to become free when it does not cause any trouble, especially in the segment of a limb having only one bone. The latter serves both as a mould and a stimulant to new ossification.

But it is better to perform early resection than to be obliged later to amputate, which will certainly happen if the periosteum is extensively destroyed. Marchant says that if we cannot stop the progress of the source of infection, if fever and symptoms of infection continue, the entire bone should be removed, or at least the diseased parts, so as to at once remove the focus of infection. In serious cases in which the entire spongy tissue of the diaphysis is infiltrated with pus, resection or total ablation of the bone should be performed.

The rule that I would give you is as follows: *Every time that the local or general symptoms are not serious, wait until there is a periosteal regeneration and then remove the sequestrum, and only then if the necrosed tissue is not eliminated by nature.*

Antisepsis must be rigidly followed, but do not employ phenol or the bichloride of mercury, both of which are badly borne by children. Eucalyptol is more powerful than boracic acid and is well borne by your little patients. The following solution recommended by Dubreuil will be found of service, and is one that I have often employed:

R Eucalyptol, gtt. x;  
Alcoholis, ʒi;  
Aq. dest., ʒii, ʒii. M.  
Sig.—Use as directed.

A one to five hundred solution of thymol is also excellent, but let me remind you not to use it during hot weather, as the patient's life will be rendered miserable by the flies, which are attracted by the strong aromatic odor of this drug.

Another point on which I would insist is the use of iodoform gauze or powder, especially in children. Personally I have not employed a single strip of this gauze for over two years, having taken to the bismuth salts, all of which can be *sterilized at 100° C.* and are non-toxic, even when freely applied to wounds in large amount, and thus you can readily conceive of their superiority over iodoform, iodol, europen, and other iodine compounds. My gauzes are made at the following strengths: subgallate of bismuth, twenty per cent.; oxyiodide of bismuth, ten per cent.; tribromophenate of bismuth, ten to fifteen per cent.; carbolate of bismuth, ten per cent. A ten-per-cent. gauze of the carbolate contains one per cent. phenic acid, while a ten-per-cent. oxyiodide gauze contains about thirty-three per cent. pure iodine and sixty-seven per cent. trioxide of bismuth. I would add that after much experience with these bismuth compounds I feel sure that clinically they are as good antiseptics as iodoform and like products, although few operators are aware of their many virtues.

The patient's limb should be done up in cotton and immobilized. You will change the dressings as often as is necessary, while the drain is to be shortened little by little as cicatrization progresses from the bottom of the wound upward.



# Gynæcology and Obstetrics.

---

## THE TREATMENT OF PLACENTA PRÆVIA.

CLINICAL LECTURE DELIVERED AT THE BAUDELLOCQUE MATERNITY.

BY A. PINARD, M.D.,

Clinical Professor of Obstetrics in the Faculty of Medicine, Paris.

---

GENTLEMEN,—Within the last two months chance has brought into our wards four women who have had during pregnancy, at varying periods, serious hemorrhages, caused by the insertion of the placenta on the lower segment of the uterus. I shall take advantage of this series of cases to treat this subject before you, and to lay down as clearly and simply as possible the best line of action for you to follow in the presence of this very serious complication. I shall use the histories of these four women as texts for my remarks; you have been able to follow the cases day by day, and have seen that in each instance, happily, the patient has recovered.

CASE I.—This woman was pregnant for the seventh time, her six other pregnancies having been normal. Her last menstrual period occurred in the first week of March, and everything passed off regularly until December 5. She was in the habit of earning her living by sewing-machine work, but had given this up completely some time previously, owing to abdominal pains from which she suffered. At this date, then, when she was in the ninth month, a slight hemorrhage occurred, but ceased spontaneously under the influence of rest.

Two days later, December 7, at half-past six in the morning, she was suddenly seized, on arising, with a violent hemorrhage. A midwife was hastily summoned, who made the diagnosis of placenta prævia, and had the patient conveyed to our clinic.

It was ten o'clock when she reached our wards, and I was making my rounds. I found her bathed in blood, but the hemorrhage had stopped. After removing a quantity of clots from the vagina we

ascertained that she was not in labor; the cervix was long and was permeable, as is always the case in multiparæ, especially when they have just had a hemorrhage. At the inner orifice it was an easy matter to feel clearly with the finger the villi of the placenta. After this, by palpation, I found the presentation to be longitudinal, with head downward, but not in the pelvis; the heart beats were normal, and the child consequently must have been alive. What was there to be done in such a situation?

The woman had not lost an excessive quantity of blood, the pulse was hardly as high as 100, the hemorrhage had stopped, and the patient's general condition was good. I decided that nothing need be done for the time being, but directed her to have hot douches, not to check the flow of blood, as this had already stopped, and such a means alone would have been insufficient, but to disinfect the vagina. I left orders, however, for the woman to be closely watched, and, if a fresh hemorrhage occurred, to rupture and make a wide opening in the membranes, and afterwards to act according to the usual method followed in my ward.

The day passed without incident, but at six in the evening, suddenly, a fresh and very abundant hemorrhage occurred. The woman had still no painful contractions, and was therefore not in labor. My head nurse inserted her hand into the vagina and two fingers into the cervix, passing directly upward, back of the symphysis, under the villi that were already detached, there she found the membranes and made a wide breach in them. But since the portion of placenta already detached came down into the cervix, and since the head was still very high up, she immediately inserted a Champetier de Ribes pouch and filled it to its maximum, in order to avoid direct detachment of any further portion of placenta by the pressure from the descending fœtus.

The hemorrhage ceased and painful contractions made their appearance. At twenty minutes after one in the early morning the pouch was expelled from the vagina. Preparations were being made to turn the child, when digital exploration showed that the head had followed the pouch and was already deeply settled in the pelvic cavity. Ten minutes later the confinement took place spontaneously, with the birth of a boy weighing seven pounds, who is to-day in fine condition and is being nursed by his mother.

Such is the very interesting history of this case. Now let us see what theoretical and practical conclusions we can derive from it.

In the first place, this abnormal mode of insertion of the placenta that produces such hemorrhages is often and usually met with in women who have had a great many children; this fact has been long known to all obstetricians and need not claim our attention any longer. Again, the hemorrhage occurred towards the end of pregnancy, which is also the rule with multiparæ who have had a number of children; with primiparæ this complication occurs at a much earlier period. Finally, the two hemorrhages of December 5 and 7 appeared insidiously, and came to an end spontaneously. These facts are not at all out of the way, and only confirm what we have already known.

A point worthier of discussion is the perception of the villi at the first digital examination made in the wards. Was the placenta inserted centre for centre, as might be thought, and, indeed, as some obstetricians do think at the present day; for my part, I have never met with this mode of insertion of the placenta, nor is the case under consideration such a one. Palpations showed very clearly that the placenta was on the posterior wall, as the head, easily felt, was in direct contact with the anterior wall of the uterus; palpation gave a very clear sensation of the head covered with the uterine wall, which was very thin and showed no interposition of placental tissue. If, in opening the membranes, the fingers had been directed backward, as the tendency usually is to do, they would have gone through the placenta. It is in this way that I explain cases of the so-called centre for centre mode of placental insertion that appears to occur so often in some wards: the placentas have had holes made in them! Instead of that, what was done in this instance? The portion of placenta that had become detached, and had gradually slipped over and in front of the inner orifice, was avoided; the two fingers of the operator passed in front, back of the symphysis, the membranes were reached and ruptured without causing any further detachment of the placental villi from the uterine wall.

The second point to be discussed, Why did I advise temporizing under close watching? Because the general condition of the patient was good, and a certain lapse of time might have passed before a recurrence of the hemorrhage occurred. This was so much clear gain for the fœtus, which, until it has gone its full nine months, develops nowhere so satisfactorily as in the uterus. Whatever may be the period of pregnancy, whether labor has set in or not, in such a case as this you have one of two situations. The patient's pulse is

either more or less than 100. If it exceed 100, take action at once; wait, on the other hand, if it is less than 100.

If the hemorrhage sets in again, as was the case with our patient, there should be no hesitation about what to do: do not plug the cervix, but make a large opening in the membranes. Later on I shall have more to say about plugging, but I shall not defer the explanation why we open the membranes freely in such cases. This is done not to bring the foetus down, but to prevent further detachment of the placenta. This is a precept to be followed even during pregnancy when there are no signs of labor, if the life of your patient is in any danger; even then a quickening of the pulse after loss of blood should be a formal indication for you to interfere in this manner. A single puncture of the enveloping membranes is quite insufficient, although this was advised in former times by Mauriceau, Puzos, etc. There is a much greater difference than is generally understood between these two methods, the puncture and the large opening made in the membranes. When a puncture is made, as soon as the liquid has run off no doubt the tension in the amniotic sac is diminished, but the chorion is still relatively intact and drags on the placenta, whereby the hemorrhage is liable to be kept up. Nothing but a free rupture of the chorion will prevent this dragging on the placenta.

But a wide opening in the membrane, however efficacious, can only act on the indirect detachment of the placenta,—that is to say, such as is affected by the traction of the chorion. It cannot alter in any respect the direct detachment that occurs when a portion of the foetus in coming down is brought to bear directly on the placenta. In the history cited above a strip of placenta had become attached to the cervix, as frequently occurs. In such a case the foetal part cannot descend without pushing the placenta before it to a certain degree, causing a most serious flow of blood. The wide opening in the membranes was an excellent remedy for the indirect detachment of the placenta, but something more is necessary for this direct detachment. This is why a Champetier de Ribes pouch, which is incompressible, became necessary, and was inserted, both to put a stop to the direct hemorrhage, to stimulate the contractions, and to effect complete dilatation without the foetal part being able to press down from above. The result of this action was, on the one hand, to stop the hemorrhage completely, and, on the other, to bring on a regular condition of labor, which was carried successfully to an end by les-

sensing or increasing the amount of liquid in the pouch according to indications.

When the pouch was driven out, if the head had not been found well down, we would have turned the fœtus in the ordinary way in order to get a rapid extraction. I am not at all a partisan of the bipolar, or Braxton-Hicks, method, which, even in the hands of its warmest advocates, all skilful operators, gives a fœtal mortality of seventy-five or eighty per cent. This method seems to me most dangerous for the fœtus, for the reason that the various manœuvres it necessitates stimulate the respiratory reflex, and in that way set up premature separation of the placenta, and cause the death of the fœtus. Furthermore, it is not applicable to all cases.

But let us return to the plugging question. I do not even refer to the use of ergot in these cases, nor to injections of ergotine or ergotinine. Although this method of medication has been very properly abandoned, such is not yet the case with plugging. I wish to speak once more of the habit of plugging, although you may think this superfluous.

The advocates of plugging still defend its use, as, they say, it forms a dam so that the blood cannot pass. This is rarely true; in almost all cases one plug has to be followed by a second, a third, a fourth, etc., and still the blood continues to flow. This is shown by the cases published by the obstetricians who favor this practice, even when it is applied by the most skilled and experienced operators.

In the second place, plugging is a difficult method to apply properly: it is very painful to the patient, and even if it fulfilled its purpose, it is at the best a very uncertain method to rely on. Suppose that no more blood does appear in the vagina, it can still accumulate behind the plug, as was found to be the case with the only woman who was ever brought to this clinic with a plug satisfactorily placed. It is therefore a blind method, as it is not directed at the cause of the hemorrhage. For this reason nearly all the children *in utero* succumb when it is applied, while their mothers are exposed to great dangers.

Finally, from an antiseptic point of view, this method is not without its drawbacks, as it is only too often a cause of infection to the patients with whom it is used. The following case is an instance in point:

CASE II.—Placenta badly placed. Profuse hemorrhages having produced a serious condition of anæmia. Patient was plugged at

her house and then sent to the hospital. After rupturing the membranes an instrumental delivery was effected. Injections of serum. Infection. Intrauterine irrigation. Curettage. Injections of anti-streptococcic serum. Recovery.

The patient is twenty-two years of age. She reached the clinic at a quarter to five in the afternoon. This is her third pregnancy. The first two, in 1893 and 1894, were conducted at this clinic, and were normal in every particular. The present one gave rise to no symptoms until a week ago, she being at the time towards the end of her seventh month.

On that day, while taking her child to a hospital, she was seized with a hemorrhage, but she continued at her occupation for three days more. On the fourth day, as the loss of blood was considerable, she sent for a midwife, who told her to remain in bed and it would stop. The hemorrhage decreased by this means, but did not completely stop. On the following day, and for the two days after, she resumed her occupation again. On the morning of the day she reached us, however, the flow of blood increased again. The patient arose, lighted the fire, and had an attack of syncope. The physician who was sent for did not come, so at noon another one was called in. He applied a plug, left a prescription, and advised her to be taken to our clinic. On her arrival here, at a quarter to five in the afternoon, in an ambulance, she was carried to the lying-in room, and her condition was then noted as follows:

The patient is bloodless and cannot articulate. She is unconscious. The pulse is filiform and 140. The extremities are cold. The corneal reflex is absent. The condition is that of a warm corpse. On examining the vagina, a lump of cotton is found the size of a walnut; it is probable that the remainder of the plug fell out when the woman was being placed in the ambulance. A portion of the detached placenta covered the orifice of the cervix. To the left the membranes were felt, much distended. They were at once freely torn open. The hemorrhage ceased immediately, and the cervix was found to be dilated to about the size of a two-franc piece; the head came well down in contact with the cervix without dragging on the placenta. An injection of ten ounces of artificial serum was also given.

The dilatation of the cervix then advanced rapidly. The cord which prolapsed when the membranes were broken no longer throbbed, and at 5.10 a dead foetus was expelled from the uterus.

Ten minutes after the first injection of serum the woman came out of the lifeless condition in which she had been brought to us at the clinic, her face began to regain its natural color, and her pulse, although still very rapid, improved in character. A second injection of seven ounces of serum was made, followed by instrumental delivery, and a long and very hot intrauterine injection. In a short time the liquid came back clear, the hard, contracted uterus could be felt above the pubis, and the hemorrhage had ceased.

A third injection of twelve ounces of serum was then given, with champagne and inhalations of oxygen. The woman was placed in her bed and surrounded with heated sheets. Pulse, then, 120; temperature, 36.2° C.

At nine in the evening twelve ounces more of the serum were given. The pulse was then 108.

Although the hemorrhage had been successfully controlled, we had subsequently to deal with another serious complication. The patient had been infected and the postpartum condition became a serious one. This, however, was also mastered by intrauterine injections, continuous irrigation, curettage, and finally by injections of antistreptococcic serum, and to-day the woman has recovered.

In this case, as will have been noted, the hemorrhage occurred not a few days before the end of pregnancy, but in the seventh month. This patient's history, therefore, illustrates very forcibly the result of plugging. During the last twenty years what a quantity of women I have seen brought into our hospitals with so-called plugs! And yet, I give you my word for it, I have seen an impassable barrier, a properly placed plug, only once, in the case of the woman to whom I referred above who died on arriving in my ward at the Lariboisiere Hospital, and who had a kilogramme (twenty-three pounds) of clots accumulated back of the plug. In all the other cases the plug was imperfectly applied. You can see for yourselves, by visiting our museum, what these plugs are, as we have made a collection of such articles taken from women who have been brought into our wards.

Now, as regards this patient, what was done the moment she reached the wards? Nothing but to make a wide breach in the membranes, and the loss of blood ceased at once. If I had made a special request for a history to justify the therapeutical means I recommend I could not have obtained a more demonstrative one. This history is, furthermore, extremely interesting and important in two other particulars.

To begin with, those of you who were in attendance in the wards when she arrived will not forget as long as you live, I feel certain, what they saw, as they were witnesses of a resurrection, due to the skill of our head nurse, whom I am happy to congratulate publicly.

The artificial serum showed what it is capable of doing on such occasions, and you were able to see for yourselves how rapid its necessity whatever of making intravenous injections, which require special apparatus and a certain amount of practice. Four injections of this serum were made in a few hours, whereby the blood-supply of the patient was reinforced by about three pints of liquid.

Although we had reason for a feeling of satisfaction at having remedied the great loss of blood, we were still not easy in our minds concerning our patient. Experience has shown us that women who have lost a large amount of blood are a favorable ground, a suitable culture-bouillon, for the development of pathogenic germs; I also know how frequently the application of a plug is synonymous with the infection of the patient. Therefore I was on the lookout for signs of infection, and unfortunately every expectation was realized.

Forty-eight hours later the first symptoms of infection appeared, and we were obliged to fight this complication for a long period. I cannot to-day go into the details of the means we employed for this purpose, nor can I now say how much credit for this woman's recovery can be ascribed to the continuous irrigation, the curettage, or the antistreptococic serum. Although I have been using this serum for some time now, I am not yet in a position to give you my opinion as to its efficacy, because when I do so I wish it to be based on a number of cases and on satisfactory comparative statistics. I wish simply to call your attention at present to the fact that this patient left the ward having had no phlegmatic alba dolens and with no lesion or induration about the uterus. I now pass to the third case.

CASE III.—Placenta badly placed. Plug applied at her home for miscarriage. Continuance of the hemorrhage. Membranes widely opened and a Champetier de Ribes pouch placed in position. Extraction of a dead foetus. Instrumental delivery.

The patient, twenty-nine years of age, reached the clinic at half-past six in the morning. This is her second pregnancy, the first having been normal in all particulars. She is now at the beginning of her sixth month, and up to the present time has not ceased her occupation as housekeeper. Seven weeks ago the first hemorrhage occurred, and continued in a slight degree for three or four days.



She kept at her work, however, used hot douches, and the flow ceased spontaneously.

Three hours before she reached us a second hemorrhage occurred, at 3.30 A.M., without warning, and was so profuse that it waked the patient up. A physician was summoned and a plug was applied. The flow stopped for half an hour, but as the blood then began to filter through the cotton, the doctor had the patient carried on a litter to our clinic.

When she was brought into the operating-room a plug, made of six lumps of cotton, was withdrawn from the vagina and a prolonged injection was made. The hemorrhage ceased. By palpation through the thick abdominal walls a breach presentation was made out in the right transverse position. The pelvis was normal; auscultation was negative; the cervix was long, and there were varicose veins and œdema of the legs especially on the left side. The fœtus was dead.

In spite of the hemorrhage the general condition was good; the pulse 80, the temperature  $37.2^{\circ}$  C., and the face presented a natural color. There were only a few and very feeble uterine contractions.

At one o'clock in the afternoon the flow reappeared, not very profuse, ceasing after a hot douche, but beginning again an hour later. Our head nurse then introduced her hand within the vaginal cavity and found a portion of the placenta detached inside the cervix; by inserting two fingers very high up to the left she finally found the membranes, but their flaccidity made their rupture a very difficult matter. When this had been effected, however, she inserted a Champetier de Ribes pouch, incompletely distended with fourteen ounces of fluid. This was at two o'clock. The pulse was then 120 and the hemorrhage had entirely stopped, but soon afterwards the patient grew pale and showed signs of syncope. She was brought to herself by the use of alcoholic drinks, and her pulse, although weak, descended to 96. Injections of normal salt solution were deemed necessary.

At 2.30 P.M. six drachms of liquid were allowed to escape from the pouch; the uterine contractions became frequent and energetic. At 4.20 the pulse was 76, and there was complete dilatation; the pouch was withdrawn. At 4.30 extraction of the fœtus by the breach was accomplished. The child, still-born, weighed two and eight-tenths pounds. Rapid delivery of the placenta was at once performed and a long intrauterine douche given. Since that time the patient's general condition has remained excellent, the pulse has

become normal, and has remained in that condition. The temperature also has been normal.

The interest in this history lies in the appearance of the hemorrhage at an early moment in pregnancy; it occurred during the fourth month, and recurred with sufficient profuseness to put a stop to the pregnancy before the end of the sixth month.

In this case you can again note the inadequate action of the plug, and you were able to see how successfully we put a stop to the hemorrhage by the use of the means with which you are familiar. Without dwelling any further on this case, I pass on to the fourth.

**CASE IV.**—The patient, thirty-one years of age, a seamstress, entered the clinic for losses of blood that had been occurring irregularly for six months. This is her second pregnancy, her first having occurred eight years ago, with a premature birth at eight months. She is now about five months pregnant.

Eighteen days after the end of her last menstrual period she was taken with a slight flow of blood, but it did not continue. The early days of this pregnancy were marked by vomiting and abdominal pain, and in the second month these pains had become so severe that the patient had been obliged to cease all work and take to her bed.

One month ago there appeared a grayish, fetid, serous discharge which ended in a week, and has since been replaced by a flow of blood varying in amount. This hemorrhage occurred both at night and in the daytime and was rather more abundant at night. During these early months of pregnancy the patient was examined by two physicians, the first made the diagnosis of abnormal pregnancy, the latter thought there was a tumor.

Two and a half weeks ago she entered a hospital, first in a medical and then in a surgical ward. In the latter the diagnosis of pregnancy was undoubtedly made, as she was advised to go home and remain constantly in bed to avoid a miscarriage. This she did for a week, but as the loss of blood continued, and her own physician feared an extrauterine pregnancy, she accepted his advice and came to our clinic.

When she entered we found the pregnant uterus coming up to the umbilicus, and made the diagnosis of pregnancy at four and a half months, the placenta being inserted on the lower segment of the uterus. The child was alive, as shown by its movements and by the foetal heart sounds. The patient's organs seemed to be in a

normal condition, but the skin and mucous membranes showed a marked loss of color. Nevertheless, as the general condition was good, and as the pulse was regular, oscillated between 70 and 80, we did not do more than place the patient in bed under careful supervision, with several hot douches *per diem*.

Twelve days later, at 5.30 in the afternoon, painful contractions of the uterus appeared, and as they became more and more intense the patient was removed to the lying-in room. At two o'clock in the night the dilatation was about the size of a two-franc piece, and the membranes, thick and stretched, could be clearly felt. Some time afterwards nothing but the placenta could be found at the orifice, but by passing high up and to the left, back of the symphysis, the breach was discovered in a left anterior position.

At 2.20 A.M. the foetus was expelled whole, the placenta foremost, there having been no hemorrhage at all during labor. The placenta, as you can see, occupies almost the whole of the membranes; in front, it covers one-third of the mass, while behind it comes up to the neck of the foetus.

A prolonged intrauterine douche was given immediately after the expulsion of the foetus, and the sequelæ of this miscarriage were perfectly normal.

The history offers the following peculiarities:

1. It proves in the clearest possible manner that when the placenta is inserted on the lower segment hemorrhage can occur even in the early months of pregnancy. In this case the flow began in the first month and continued more or less incessantly for five months.

2. It brings to mind the cases published by Simpson, in which the placenta came foremost without there being any hemorrhage during labor.

3. Lastly, it proves in the most positive way that the placenta can slide over the orifice during the period of dilatation, since, at the beginning of labor nothing but the membranes could be felt, and it was not until the dilatation was greater than the size of a two-franc piece that the edge of the placenta was felt. Yet, in examining the whole mass, as I show it to you now, it would be difficult not to believe in a central insertion of the placenta.

You see, consequently, how uncertain is the value of the examination of placenta, after delivery, with regard to the insertion of the placenta, and I need dwell on this fact no further to-day.

I wish to call your attention to one point. In the case of the

woman whose history you have just heard the hemorrhage was at no time serious in quantity, but only became dangerous by its persistency. I had the patient closely watched, and would have interrupted the pregnancy at any moment if her pulse had gone up to 100.

I shall now close by giving you a *résumé* of the line of action that I recommend:

1. Whenever during pregnancy or labor a hemorrhage due to a badly inserted placenta is sufficiently serious to raise the pulse above 100, you must act promptly.

2. Your action must aim at removing the cause of the hemorrhage and at hastening so far as possible the expulsion of the fœtus. A wide opening in the membranes will put an end to the first cause of the hemorrhage, and is in itself generally sufficient. The placing of an incompressible pouch within the cervix will put a stop to the second cause of danger,—that is to say, will prevent the fœtus from directly detaching the placenta while coming down, and will hasten the labor.

3. When, as often happens, a hemorrhage occurs during delivery, either before or immediately after the expulsion or the extraction of the placenta, give prolonged, intrauterine douches of hot water at 48° to 50° C.

4. When the quantity of blood lost has been considerable, and has caused signs of serious, acute anæmia, use injections of normal salt solution (i.e., artificial serum), given in the cellular tissue, at a dose varying from seven to seventy ounces, according to the gravity of the case.

By following these precepts rigorously we have obtained the results with which you are familiar, and when I compare these results with those obtained by other obstetricians using different means, I think I have the right, and that it is my duty, to tell you that at the present time I know of no better therapeutical means than that I have advised you to use.

**EXCESSIVE MOBILITY OF UTERUS; PELVIC PERITONITIS AND FIBROID TUMOR; RETROFLEXION AND PROLAPSED OVARY; LACERATIONS AND CICATRICES OF VAGINA; AFTER-TREATMENT OF TRACHELORRHAPHY; RETROVERSION OF THE PREGNANT UTERUS.**

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY PAUL F. MUNDE, M.D., LL.D.,

Professor of Gynecology at the New York Polyclinic and at Dartmouth College;  
Gynecologist to Mt. Sinai Hospital, New York City, New York.

---

**EXCESSIVE MOBILITY OF THE UTERUS.**

GENTLEMEN,—This patient is a married woman who has had several children, the last one two years ago. Menstruation is regular and normal. She comes to us complaining of pain in the lower part of the back and of a profuse vaginal discharge. Besides the fact that the normal ovaries can be so readily felt here, which is partly owing to the great relaxation of the abdominal walls, the excessive mobility of the uterus is a point worthy of note. The uterus in its normal position is movable, and there are several degrees of normal mobility of the uterus. With this organ in its normal position and the ligaments in their normal condition, the uterus should be so movable that it can be moved forward one degree, backward two degrees, and laterally one degree, and as soon as the pressure is relaxed it should resume its original position. When this normal mobility is lessened, it is probable that there has been some inflammation which has caused the pelvic ligaments and the pelvic floor to be more rigid than they should be. If the uterus be abnormally movable it is probably a congenital condition, or it is due to relaxation and imperfect involution following frequent parturition. This is probably the cause of the extreme mobility in this patient. The cervix can be made to touch the symphysis pubis or the sacrum; it can be pushed from side to side, or downward until the cervix touches the floor of the

pelvis, and it does not resume its former position as it should do. This simply shows that the pelvic ligaments are all much relaxed. I have known of cases in which the woman would complain of the feeling of mobility, and on examination, particularly if made with the patient in an erect position, I would find this condition of excessive mobility present. This free mobility is not very common, but when it gives rise to discomfort, it calls for treatment. I employ for this purpose the ordinary flexible ring pessary known as the Meigs, or the Peaslee ring. It is made out of a spiral watch-spring covered with soft rubber, and becomes quickly offensive unless the patient removes and cleans it at least once a week. If the uterus shows a decided tendency to drop forward, you will find the flexible pessary, known as the Hitchcock, a useful one. This is also made of soft rubber, and has a projecting bow which comes in front of the cervix.

#### PELVIC PERITONITIS AND FIBROID TUMOR.

Our next patient says that she has been married two years, and has had one child, four months ago. Her last menstruation was one week ago. She complains of pain in the back and right side of the abdomen.

This woman came to the hospital with a history of pelvic inflammation. She had a considerable temperature and complained of much pain on the right side, and on examination I found a hard, intraperitoneal exudation on the right side of the pelvis, which rendered the uterus immovable, but it differed in no way from the exudations usually found in cases of pelvic peritonitis. After an application of ice for two weeks, or until the temperature was controlled, she was treated by hot applications and blisters, and remained in the hospital altogether for a period of seven weeks. An examination made at the end of that time showed that the exudation had almost entirely disappeared, but the uterus was still bound down by adhesions, and that, in addition to this, there was a tumor on the right side of the uterus, and apparently attached to it, just above the insertion of the vagina. This tumor was about the size of the fist, fairly movable, and quite hard. It was thought to be a subperitoneal fibroid which had been present before the attack of pelvic peritonitis. The tumor now feels smaller than it did just before her leaving the hospital, four weeks ago, and this makes me a little doubtful as to the correctness of the former diagnosis, for it may have been simply a hard

exudate. You may say, Why can you not tell by the palpation of the ovary? This would settle the difficulty in many instances, but in cases of pelvic peritonitis the pelvic organs are apt to be covered by exudate, so that the fibroid might coexist and yet not be detected. The interesting point about this patient can best be made clear to you by an illustrative case.

Some years ago I was called in consultation to see a lady who had a temperature of  $102^{\circ}$  F. and a pulse of 100, and who was suffering much pain in the lower part of the abdomen. She had been sick in bed for two weeks. On examining her, I found the uterus pushed up towards the anterior abdominal wall and behind it was a large tumor filling out the posterior portion of the pelvic cavity, hard, immovable, and quite tender to the touch. The body of the uterus could not be felt. I diagnosticated pelvic peritonitis with a large exudate in Douglas's pouch, and I advised the usual treatment by poultices and hot douches, and gave the usual favorable prognosis. About four months later the patient called at my office and requested an examination, to be sure that she was entirely well. On examining her I found, to my great surprise, exactly the condition found here in this patient: the same hard mass that I had felt four months previous. But it was no longer tender, and there was no fever. A moment's reflection showed me that it was a case of old subperitoneal fibroid, which had become covered with an inflammatory exudate sufficient to mask the true relation of the mass to the uterus, and that this having nearly disappeared, the actual condition was revealed. I took her to Dr. Thomas, who confirmed this diagnosis. It is therefore well to bear in mind that fibroids may coexist with pelvic peritonitis, and may be present after the disappearance of the exudation.

#### RETROFLEXION AND PROLAPSED OVARY.

I would next call your attention to this patient, thirty-one years old, who has been married nine years, and has had five children, the last about six months ago. She now comes complaining of pain in the back and left side of the abdomen, which she has had for three years. She is nursing now, but before this pregnancy she menstruated profusely, flowing every two or three weeks for a period of six or seven days. There is also present a very marked sticky, white vaginal discharge.

Inspection shows the perineum torn half-way to the sphincter, and there is a protrusion of the posterior vaginal wall, which comes

from laceration of the perineum and subinvolution of the posterior vaginal wall. I have already shown you a number of such cases. The examining finger shows the cervix to be slightly torn, but immediately behind the cervix my finger encounters the body of the uterus. Between the cervix and this mass is a furrow which can be indented with the finger. We have here a retroflexion of the uterus. The body of the uterus can be readily moved, and it is important that we should determine this fact in order to be able to treat the case intelligently. On passing the finger farther up towards the fundus it encounters a small and very tender body, which is irregular, nodular, and about the size of a large almond. It is the left ovary, but as it is movable it will probably not complicate the case. The diagnosis in this patient is laceration of the perineum, rectocele, prolapse of the uterus in the first degree, retroflexion of the uterus, and prolapse of the left ovary. The pelvic organs have not yet returned to their normal, antepregnant condition, the uterus being large and the vaginal orifice gaping. Ordinarily, involution is completed within two months, but there are many cases in which subinvolution lasts for a year or two. If the cervix be torn or the uterus out of place, the circulation is interfered with, and therefore the process of involution is imperfect. By the use of proper supports for the uterus, aided by hot douches, a decided improvement should soon be noticed. The pain in her back is due to the low and backward position of the uterus, and the pain in the left side probably arises from the prolapse of the ovary and the consequent congestion of this organ. In addition to congestion, if the patient be constipated, the hard fæcal matter passes along through the sigmoid flexure directly over the tender ovary. The indications here are to replace the uterus and ovary, and then to keep both in position. The uterus is usually replaced with the finger, but the ovary is very liable to drop back again, hence it may be necessary to place the woman in that position in which the ovary will slip back of itself,—namely, the knee-chest position,—and then, by lifting up the perineum with the finger or speculum, the uterus and ovaries will be replaced almost by themselves, partly by the action of gravity and partly by the atmospheric pressure. Dr. Sims used to declare that the inflation of the vagina with air gave rise to a force equal to twenty pounds to the square inch. While I think this exact estimate of force needs further corroboration, it is quite likely that this renders valuable assistance. I have seen an impacted uterus replaced



by putting the woman in the knee-chest position, and simply pulling back the perineum. Now if the uterus be retroverted or retroflexed, place the patient in the ordinary Sims position and stand behind her with your foot upon a stool, so that your right elbow may be conveniently steadied by resting it upon your knee, then insert two fingers into the vagina and push the body of the uterus upward, pressing behind the cervix until the middle finger no longer reaches the body of the uterus, then, keeping the middle finger still behind the cervix, catch the cervix in front with the index-finger, and so tilt the fundus uteri still farther forward until it is somewhat anteverted. Now introduce the supporter which you have selected. I have just done this in the case before us, and in order to demonstrate to you all that the uterus is now in its proper position, I introduce the sound into the uterus. When making a digital examination with the patient in the Sims position, Barnes recommends the use of the left hand, but this I consider very awkward. It is often necessary to introduce tannin and iodoform tampons until the sensitiveness has disappeared sufficiently to allow the use of a pessary. This patient requires a pretty large and sharply curved Hodge pessary, one which is nearly as broad in front as it is behind; otherwise it will not retain its position. Dr. Noeggerath's pessary, which is similar, is good for cases of relaxed vagina and perineum. Sometimes when the ovary is prolapsed and it is difficult to keep it replaced, I employ one with a large, thick posterior bar.

Of course, the treatment outlined will not be sufficient to *cure* the case. I have cured very few cases with pessaries. The reported cures are usually those in which the uterus has been found in place when the pessary has been worn for a few months, and where no subsequent examination has been made. If you remove a splint from a broken leg or a dislocated arm, and the limb is not moved, of course the position of the part will be excellent; but that does not mean that a cure has been effected. It is only in recent cases, and when the treatment is begun very soon after confinement, that in my experience a cure by a pessary is to be expected. If a cure were demanded here, I might shorten the round ligaments by Alexander's operation, or perform an anterior ventral fixation, and sew up the vagina and perineum by some plastic operation; but the majority of women will be satisfied if they can be relieved by a pessary. Pessaries after all are only make-shifts, but they are excellent within certain limits.

## LACERATIONS AND CICATRICES OF THE VAGINA.

This patient is forty years of age, has been married twenty-three years, and has had eight children, the last one about one year ago. She is unwell every four or six weeks, the flow being normal in quantity, and the last time three weeks ago. She complains of pain on the left side, and of a rather profuse white vaginal discharge. There is a slight perineal laceration, and a laceration of the cervix extending up into the right vaginal vault, which probably has previously extended into the walls of the vagina. This has cicatrized, leaving two cicatricial folds in the right vaginal vault. These peculiar cicatricial bands do at times cause some discomfort. A week ago I saw an elderly lady who came to me for dyspareunia, and on examination I found nothing abnormal except that the posterior vaginal vault was shortened and there were two cicatricial bands extending from the cervix down the posterior vaginal wall. This lady was long past the menopause, and these cicatricial bands were simply the result of the normal senile contraction; the cervix had shrunk and the vagina had become somewhat shortened, and in this process the muscular fibres of the vagina had assumed the character of the bands already described. The pain experienced on intercourse was exactly localized at these bands. The treatment of such cases consists in taking sharp-pointed scissors, and with the aid of the Sims speculum, snipping these bands superficially in several places, thus enabling the finger to break them down and restore the normal size of the vagina. Deep incisions are liable to cause severe hemorrhage, or, if done behind the cervix, may even open Douglas's cul-de-sac. These cicatricial bands are by no means uncommon, and are due to lacerations of the cervix or of the vagina, usually as the result of childbirth. I have seen lacerations of the cervix extending from the cervix up into the vaginal vault, so that a pelvic cellulitis was produced, and subsequently an abscess, which kept the patient in bed for a long time. Such lacerations are usually the result of forceps operations performed before the cervix was thoroughly dilated, and the extraction too rapidly done to admit of proper yielding of the cervical tissues. But childbirth is not the only cause. I have seen two cases of most severe laceration of the vaginal wall produced by a first connection. In one case the laceration extended from the hymen up the left side fully two inches into the vagina,

and the woman had lost so much blood that she became collapsed, and it was only by very rapidly emptying the vagina of the coagula which distended it and making pressure by tampons that the hemorrhage was controlled. In the second case, the laceration extended from the vaginal vault two or three inches down the vagina, but did not involve the hymen at all. She also went into collapse, and the vaginal walls were not only torn, but were literally crushed. An examination of the male organ in this case did not indicate any undue disproportion in size of the male and female organs. It is not at all uncommon for women who have passed the menopause, and where the tissues have become quite brittle, to suffer from severe lacerations of the vagina upon resuming sexual intercourse after a long period of abstinence.

#### AFTER-TREATMENT OF TRACHELORRHAPHY.

Independently of the history of a case, it is sometimes desirable to know whether or not a patient has borne children, and ordinarily the existence of the hymen, or of a large portion of it, will negative this question. Its absence, however, does not necessarily mean that the woman has had a child, for she may have been delivered of a large tumor. In this next patient, the hymen still persists, although she has had one child. Usually the posterior portion is entirely destroyed by the sloughing away of the *carunculae myrtiformes*. On examination of this patient, I find that a trachelorrhaphy has been performed upon her, and the stitches are still in the cervix. I shall use this case as a text for a few remarks on the after-treatment of patients subjected to this operation. For about ten days after the operation the patient is kept in bed, and not allowed even to sit up. This may seem to be unnecessary caution, but when we consider the dragging down of the uterus which occurs during the operation this period of rest seems only prudent, even though the healing process were proceeding satisfactorily. During this period, carbolyzed douches are employed to preserve cleanliness, the bowels are kept regular, and she is allowed to pass her urine voluntarily if possible. After any operation, the catheter should be avoided as much as possible, for its use is very prone to cause irritation of the bladder, which may easily prove more annoying than the operation itself. I do not allow the catheter to be passed by touch alone, but insist that the urethra and adjoining parts be thoroughly cleansed and then

the catheter introduced by sight. The usual custom is to remove the sutures at the end of about ten days, but my habit for sometime past has been to examine the patient with the Sims speculum at the end of this time, and if the stitches do not appear to be causing irritation, or are not in danger of cutting through, I leave them alone, and allow the patient first to sit up, and then to walk around. If she menstruates within a short time, I leave the stitches in until this is past. After this they are removed, the vagina cleansed, and a tampon of tannin and iodoform introduced. This completes the treatment. This practice has not only done no harm, but has secured better healing. Of course, coition should always be interdicted in any case for at least a month afterwards. Occasionally a suture is inadvertently allowed to remain, but such an occurrence usually leads to no complaint except on the part of the husband. Formerly, I used to remove the stitches about the tenth day, but several years ago it happened that, in two or three cases, after the removal of the stitches at this time, I wiped the parts with cotton, and then, on looking in again, the whole surface was found separated. I sewed up the cervix at once, and allowed these patients to go around for about ten days more, when examination showed that the parts were thoroughly healed. As regards the manner of removing the sutures, it is only necessary to say that the cervix is exposed with the Sims speculum, and with an ordinary uterine dressing forceps, the stitch farthest away from the external os is grasped, and the suture cut, care being taken not to cut off the twist, for then it is almost impossible to find the suture. If the nearest suture be removed first, one is likely to tear open the cervix in removing the other less accessible ones. If the upper ones be removed first, and there should be a little oozing, the field of operation is obscured by the blood. After removing the stitches, the sound is introduced to be sure that there is no obstruction in the cervical canal. The fissures left by the sutures will usually be obliterated in about a week. The operation of trachelorrhaphy often fails when done for the relief of pelvic pain, but I can point to hundreds of cases which I have operated upon in the last fifteen years, who were leading a miserable existence before the operation, and yet have been well since that time. If you only operate when the symptoms plainly indicate that they are caused by the tear, then, I think, you will not be disappointed in the results of this operation.

## RETROVERSION OF THE PREGNANT UTERUS.

Our next patient is thirty-two years of age, has been married five years, and has had two children, and one premature labor at seven months, and a miscarriage six months ago. She was last unwell thirteen weeks ago, and came here seven weeks ago on account of backache and bearing-down pain. The examination at that time showed the uterus enlarged and soft, and turned backward, and a diagnosis was made of pregnancy with retroversion. These cases of retroversion of the gravid uterus are of considerable interest. In the first place the question arises, Does the backward displacement precede or follow conception? As a rule, a woman with a retroversion or particularly a retroflexion is not so liable to conceive as one with a uterus in the normal position, but of course it not infrequently occurs. The uterus lying in the hollow of the sacrum will not replace itself, for the promontory of the sacrum will interfere with the normal rising of the uterus into the abdominal cavity, and as a result impaction of the uterus, as it is called, takes place; then labor-pains occur, and finally abortion. If the uterus were only slightly tipped backward, and pregnancy occurred, probably about the third month a slight jar would tip the uterus still farther backward. I have seen this occur in cases which had been under my observation prior to this accident. Unfortunately we are not aware in most cases of the existence of impaction until symptoms have appeared, and hence, it is often too late to save the fœtus. However, I have seen cases where the pains were very severe, and where I had difficulty in replacing the uterus, and yet miscarriage was averted. In this patient we have introduced a pessary to prevent such an accident.

Having made a correct diagnosis, the first thing to do is to replace the uterus. This can be done by putting the woman on the left side and pushing the fundus upward with two fingers, and drawing the cervix back with the index-finger when the fundus has almost passed out of reach. In bad cases of impaction there is so much tenderness that you cannot use so much pressure, for fear of causing an abortion; and then the patient should be put in the knee-chest position, a Sims speculum introduced, and the cervix exposed. The mere action of drawing back the perineum in this way will sometimes cause the replacement of the uterus, and it was in this way that the uterus was replaced in this patient before the introduction

of the pessary. If this spontaneous reposition does not occur, hook a tenaculum into the anterior lip of the cervix and pull the uterus towards the entrance of the vagina, and then with the same tenaculum carry it backward towards the promontory of the sacrum. This manœuvre will almost always succeed, and it is much pleasanter and safer than the old method of introducing two fingers into the rectum. In very bad cases it has been recommended to insert a rubber colpeurynter into the rectum, and fill it with water, so as to exert steady pressure upon the uterus. It is undoubtedly a very excellent method, but it is rarely necessary. I have sometimes introduced the ordinary vaginal depressor into the rectum, and so exerted pressure upon the uterus. Having replaced the uterus while the woman is still in this position, a properly fitting pessary is inserted, and the patient kept quiet for a day or two. The next question is, How long shall the pessary be allowed to remain? There is no occasion to leave it in after the fourth month, because the fundus is above the promontory of the sacrum and the uterus cannot then become retroverted, hence, about the beginning of the fourth month it should be removed. If left longer, besides being unnecessary, it would cause undue irritation and pressure. The subsequent course of such a case will probably be perfectly normal, but when the patient gets up after confinement, if the retroversion existed before conception, it is very probable that it will return, and then is the time to cure the displacement by means of a pessary. When the uterus and its ligaments are still relaxed and are contracting by natural efforts is the proper time, before the patient is out of bed, to introduce the pessary, having made certain that the uterus is in proper position. Let her wear it for from three to six months, and if the displacement is to be cured by such a support, this is the time when it will prove successful. I do not think pessaries often *cure* displacements, although I am aware that other gentlemen claim much better results from the use of these instruments. It must be remembered that, although the uterus be in position when the pessary is removed, the displacement may very quickly recur. The cases I have cured have been mostly after confinements or operations, or where the displacement was of recent origin. In a few cases, as a result of impaction of the pregnant uterus, the pressure of the cervix upon the vagina and bladder has been so excessive as to cause mechanical retention of urine, and, being unrelieved for some time, has resulted in gangrene of the entire mucous membrane of the bladder.

# EPITHELIOMA OF THE VULVA; RETROPOSITION OF THE UTERUS.

CLINICAL LECTURE DELIVERED AT THE JEFFERSON HOSPITAL.

BY E. E. MONTGOMERY, M.D.,

Professor of Clinical Gynæcology in the Jefferson Medical College; Gynæcologist to Jefferson and St. Joseph's Hospitals; President of the Philadelphia Obstetrical Society; Ex-President of the Pennsylvania State Medical Society, etc., Philadelphia, Pennsylvania.

---

GENTLEMEN,—This patient, forty-seven years of age, with father living, has lost her mother with pneumonia. Three brothers are in good health and four sisters have died. There is no history of malignant disease. She has always enjoyed good health until her present trouble. She was married when twenty-eight years old, has given birth to five children, and has never had a miscarriage. Her labors were normal and the convalescence undisturbed. The menopause occurred three years since. About that time she first noticed a small swelling, a tumor, in the right labium minor. This was not painful, but slightly increased in size until a year ago, when it began to ulcerate and became exceedingly painful. Since then it has steadily grown worse. She has lost weight during the last year, and suffers constant pain except when under the influence of opium. Upon examination we find one-half of the right labium destroyed. This ulceration presents a raw, ragged surface, which extends upward; the ulceration has destroyed the smaller labium and the clitoris, and extends on the left side over the upper half of the labium, so that both labia minora are destroyed. As we look at the surface we see a ragged edge, an ulcerated surface, which is not exposed until the labia are separated. It becomes an interesting point to determine the character of this condition. She is forty-seven years old, and this trouble began three years ago as a mass in the right labium, which has subsequently increased in size, undergone ulceration, and caused considerable destruction of tissue. You will notice that the

surface is roughened, with the appearance at points of partial extravasation, and has a hard, firm base. There are a number of possibilities which must be considered in arriving at a diagnosis. It may be a syphilitic manifestation, a tubercular ulceration, or an epithelioma. These three diseases are the ones which would most likely be considered, and should be kept in mind in arriving at a diagnosis. In reviewing the history, we have nothing to indicate that this patient has experienced any of the ordinary symptoms of primary or secondary syphilis. The length of time the disease has existed precludes the probability of it having had such a cause. We find no such extensive ulceration in syphilis, and so we exclude that disease. Now as regards tubercular disease, we may have tubercular infection about the vulva, vagina, urethra, uterus, Fallopian tubes,—in fact, any part of the genital organs may be subject to tubercular disease. Tubercular trouble in such cases usually presents a similar appearance to lupus of the skin. As it increases, it follows a kind of serpentine course in which there will be destruction of the tissue, with a partial repair following, so that where there is a large amount of tissue destroyed there will be cicatricial tissue behind it, indicating that nature has recovered her forces in places; but as we look at this we find that it presents no such appearance. Here is a trouble which began three years since, in which the ulceration has existed for a year, and this ulceration is everywhere extending without the slightest indication of nature's ability to repair the process. This history, then, excludes the possibility of tuberculosis. From a macroscopical examination of the tissues, we are forced to the conclusion that we have to deal with epithelioma. We have no doubt that a microscopical examination of a section of this tissue would confirm the diagnosis. This diagnosis is still further confirmed by development of trouble in the lymphatic glands of the groin. These glands are infiltrated, and I have no doubt that other and deeper glands are also involved. While the patient is suffering from the disordered condition which would seem to me to justify us in making an effort to give her some relief, she has an extensive mass of ulceration involving the greater part of the vulva, over which the urine flows, giving rise to considerable distress and discomfort, in addition to the annoyance from the offensive discharge. Under such circumstances we have to decide what is best to do. Shall we let her go on with a condition of this kind until her life is destroyed by it, or shall we make an effort to remove



the disease, even though it is probable there will be a redevelopment of the trouble? We decided to do the operation with a view of making her more comfortable. Now, in doing this operation we got beyond the disease as far as possible, and in order so to do it was necessary to remove the greater part of both labia majora and the labia minora, the glans clitoridis having been already lost. So an incision was made over the right labium outside the disease which we extended into the mons veneris, cutting down to the pubic bone. The crura of the clitoris, bulbs of the vestibule, a portion of the vestibule itself, and the orifice of the urethra were removed. It is true that the orifice of the urethra itself was not involved, the disease having extended up to the urethra, leaving it apparently uninvolved. To make sure, however, that all the disease is removed, it was necessary to amputate the urethra, stitching its mucous membrane back to that of the vagina posteriorly and to the skin anteriorly, which was brought together on either side with sutures, so that we used what remained of the labium to close the wound. This operation was done four days ago. As I turn the patient about you see the result. There is very little disfiguration so far as the external appearance is concerned, and the orifice of the vagina and urethra are in contact with the skin. The labia are closed above down to the orifice of the urethra and the skin is stitched to the surface of the urethra itself. Malignant disease of the vulva occurs much more rarely than it does in the uterus, and still less in the vagina than in the vulva. We do, however, see, and particularly in public clinics like this, cases of malignant disease involving the labia, not usually so extensive as in this particular instance. If the patient had been seen earlier, when the disease only involved one or other labium, the operation would consist in the amputation of the labium, getting well beyond the diseased tissue, leaving, of course, in such a case no tissue that is infiltrated by the disease. In this patient we can give no hope of absolute relief, when the involvement is so extensive as to require both groins to be opened and several of the lymphatic glands to be removed. No doubt other deeper ones are involved which will subsequently cause trouble. It is important that malignant disease should be early recognized and not permitted to go on for three years, as has been done in this patient, before resorting to radical treatment. Had this condition been recognized in the first six months or possibly the first year of its development, before breaking down took place, with glandular involvement, the

chances for return to health would be fairly good, but at this time all we can do is to palliate the condition, remove the diseased tissue as far as possible, and make her comfortable for a longer period.

#### RETROPOSITION OF THE UTERUS.

The next patient is thirty-two years of age, has been married four and one-half years, and had one child three and one-half years ago, in which the labor and puerperium were apparently normal. Her menses are regular. Since the birth of her child she complains of continuous pain in the back and also at times in the right side. Upon examination we find a retrodisplacement of the uterus; in other words, the uterus is retroposed without being either retroverted or flexed. This condition is undoubtedly due to inflammation which has either followed her confinement or been the result of some other cause. Bands of adhesions are formed which draw the uterus back. This not infrequently takes place as a result of infiltration of the utero-sacral ligaments and is not always accompanied with involvement of the tubes or ovaries. In such a patient it is better that she should be subjected to treatment to bring about relief without operative interference. I have had no opportunity to investigate her condition since the first examination. I shall make a digital examination to ascertain her condition, an outline of which I will give you later. As I place the hand over the abdomen and a finger in the vagina behind the uterus, I find the latter can be drawn forward and is quite movable, can be drawn towards the symphysis; that there is some thickening in the broad ligament on the right side, not so marked as when the patient was first under observation. The uterus, considering the trouble she has had, is very fairly movable. Taking a patient who is suffering from inflammation of the uterus, in which the organ is more or less fixed in the pelvis, generally more frequently displaced backward, in a state of retroversion or retroflexion, an examination fails to disclose the presence of any accumulation in the tube or extensive inflammation of the ovary. Such patients will not infrequently complain of distress and discomfort resulting from interference with the circulation, and the organs being more or less fixed, increased distress during coition and in locomotion. The condition may be so marked as to demand interference to secure relief. I suppose there is no class of cases which demand more consideration than these of retrodisplacement of the uterus. You can readily understand that the patient in such a condition,

with a retroposed organ, in which there is more or less fixation, would suffer distress rather than receive comfort from the introduction of a pessary. The mechanical means, or the use of the pessary, only aggravates rather than gives relief. With such fixation of these organs, injections of hot water, the introduction of vaginal tampons and cotton, making pressure against the uterus, and so medicated as to deplete it, affords relief. These methods of treatment have a certain amount of influence in lessening the congestion of the organ, and raising it up improves the circulation, but they do not accomplish a complete cure. The pressure of the tampon against the uterus has no influence upon the adhesions in its upper part; it simply lifts the entire organ up, adhesions and all, without producing any special effect or influence upon the exudate. Consequently, we have to consider what are the other means to which we may resort for relief. Shall we subject such a patient to the removal of ovaries and tubes in order to afford relief? Certainly not. If we can by any means save these organs rather than sacrifice them, that should be our plan of procedure. We have in the practice of pelvic massage a method of treatment which is applicable to such cases which will in many instances remove the necessity of operative interference, which will enable the patient to recover the normal functions of her organs, and will secure the entire removal of the pelvic exudate. Of course, if these patients are seen early in the trouble, in the beginning of the acute attack, we should not resort to such measures. Where there is considerable exudate the proper plan would be to make an incision through the pelvic peritoneum by way of the vagina, break up the mass of exudation and adhesions, pack the cavity with gauze, and thus afford a drain or vent for the disappearance or discharge of the exudate. This plan of procedure will afford an efficient method of controlling the disease if performed early. Breaking up the exudate in the broad ligaments and the introduction of gauze will save months of treatment, suffering, and distress, oftentimes save the patient the necessity of undergoing a sacrificial operation. But we see the patient after this acute period is passed, when she is suffering from the sequelæ of the inflammation. Our plan of procedure in such a case would be pelvic massage two or three times a week, placing one or two fingers in the vagina, pushing up the uterus against the abdominal wall, while the external hand upon the latter makes a gentle rotary motion, increasing the force gradually until we are able to

make considerable pressure. This motion is then supplemented by certain movements on the surface, picking the uterus up between the fingers and drawing a number of times upward and downward, dragging upon the exudate and the ligaments filled with it, and promoting increased elasticity of the parts. The increased activity of the circulation will secure more rapid removal of the exudate. In this way we secure the same influence and effect as we would by the passive motion of an ankylosed joint or by placing a joint at rest for a time after injury. The uterus is moved upward and downward, from side to side, in such a way as to make traction on all the bands of adhesion, and if this is pursued from day to day, following the massage with the introduction of tampons saturated with glycerine to keep the uterus elevated and deplete the parts, with the simultaneous internal administration of tonics, we will not infrequently be able to bring about a removal of the excessive amount of exudate and set a uterus free which has been long fixed, and avoid the necessity for an operation. This plan of treatment is regarded very much askance, as yet, by American physicians. You will find that if such a plan is up for discussion before one of our medical societies, there will be some one who will pronounce it a violation of ethics to practise such treatment. One man has said that it is refined masturbation; but in the method I have suggested there is no necessity for any such consideration of it, as it produces no such abnormal effect upon the individual. It can be practised with relief of the patient, and is too valuable a procedure to be thrown away without proper consideration. If we had examined this patient and found situated upon either side a mass which gave rise to a sensation of fluctuation, in which there was probably an accumulation of pus or serum or blood, what should then have been our course of treatment? Is it necessary in such a patient that the individual should undergo a radical operation? Not at all. Even the presence of such an accumulation may be treated by opening the broad ligament if necessary, exposing the pus-sac, opening this, draining, irrigating, breaking up the sac, and finally packing it with gauze. This gauze packing should be repeated until we are sure the cavity is filled by granulations, keeping the vaginal opening free. The recovery from the local condition may be followed by massage, as already directed. This subject of pelvic inflammation is one of the most important. If we find upon examination of a case that there is infiltration upon both sides, probably pus collections of extensive character, so exten-

sive as to preclude the probability of restoration of function of the parts, we have then to consider the necessity of operative interference. We certainly would not retain organs which are likely to give rise to a state of ill-health, which would constantly keep the individual an invalid. In such a case we should, of course, resort to operation for the removal of the diseased tissue. This should be done in that way which will afford the best opportunity for complete relief. If the condition is one in which the disease has existed for a length of time, in which there are evidently old firm adhesions, in which everything is firmly matted together and immovable, the pelvis presenting a hard, board-like appearance, the operation will be most readily done through the abdominal cavity, because we are better able by that course to see the parts through which the operation is to be made, to break up the adhesions under the sight, and treat the conditions that are present. If the condition, however, is a more recent one, in which there are evidently pus collections, in which it is demonstrable that the mass has been in a state of recent inflammation, the vaginal procedure is preferable; and this is also the proper course where the collection of pus is a large one, for the reason that we are better able to drain the pus cavity, evacuate it, and cleanse the surface before the peritoneal cavity is opened. We open into the pus cavity first upon one side and then upon the other, thoroughly irrigating and cleansing it, using for this purpose hydrozone, followed by sterilized water, or a one to one thousand solution of formalin. After the parts are cleansed we may then open into the peritoneal cavity, amputate the cervix, turn down the fundus of the uterus, separating the adhesions, removing the fundus, and, if necessary, subsequently following it by removal of the ovaries and tubes. In cases in which the organ is with difficulty brought down, we may apply small forceps temporarily upon the broad ligaments, removing the uterus, and then turning down the ovary and tube, for which more room is thus afforded, and apply a large pair of forceps on that portion of the broad ligament, taking away the smaller ones. In this way we may completely clean out the pelvis, and the advantage it has over the abdominal procedure is, the operation has been done with less disturbance of the general peritoneum, with no opportunity for its infection or soiling by the passing through it of a large quantity of pus. Further, the large opening in the most dependent portion of the pelvis gives better opportunity for drainage. By our gauze packing we practically

place the general peritoneal cavity in such a position that it is quickly shut off from the opportunity of infection, and the only portion with which we have to deal is that within the pelvis, which is readily reached through the vagina. In addition to this, we have another advantage in that the vessels which pass through the tissue, which has been more or less infected, are secured by forceps and not by ligature. These vessels, secured by forceps, are under ready control, and we consequently have no ligature to remain to be infected and give rise to sinus, irritation, and suppuration until the ligature comes away in a month, six months, or two years, as frequently is the case. In addition to this we have the patient with no external wound, which is a matter of considerable consideration where the patient is nervous, and we avoid the possibility of any of the sequelæ from an external wound, such as stitch abscesses or ventral hernia. These, then, are the various advantages derived from the vaginal method rather than the abdominal removal of inflammatory collections. But the most important consideration in the treatment of such cases is that of prophylaxis; the prevention of inflammations extending to the destruction of the tissues is as important a measure as any we have under review. These troubles arise most generally as a result of sepsis following abortion, miscarriage, or from traumatism the result of the introduction of instruments in the uterus, or, again, from the occurrence of gonorrhœa. It was formerly the habit to assert that gonorrhœa was the cause of the great majority of such conditions. This is not true. While gonorrhœa does prepare a favorable soil for the streptococcus infection, it is not so frequently the cause of infection as is careless treatment, extension of inflammation following delivery of the fœtus, either an abortion or delivery at term. When we have a patient suffering from inflammation of the uterus and the development of endometritis, whether it be septic in character or result from gonorrhœa, or whether it be a chronic inflammation which has existed for a length of time, our treatment should be directed to its relief as early as possible, rather than to permit the trouble to go on to the development of inflammation in structures not so readily reached. The most important consideration in such cases is to render the uterine canal sterile as quickly as possible and thus prevent the further extension of disease; not only to render the uterine canal sterile, but also to promote and increase the ease of its drainage. Inflammation in the uterus is just like inflammation in a sinus. Drainage is insufficient. Decreased ability of the dis-

charge to get out of the uterus results in that organ becoming contracted in order to force the evacuation, which is likely to cause a regurgitation into the tubes, and we have a development of trouble in these which may pass into the peritoneal cavity. So it is important to bring about as early as possible the cleansing of the uterine cavity, the promotion of a healthy condition within it before the disease extends from the uterus to the tubes and deeper pelvic structures. An exudate from tubal inflammation of a slight character does not necessarily indicate that the abdomen should be opened. Even in such cases, dilatation and curettement of the uterus with improved drainage of that organ will in many cases bring about a resolution of inflammation in the tubes and relief of the patient.

## UTERINE HEMORRHAGE.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY HENRY C. COE, M.D.,

Professor of Clinical Gynæcology in the Bellevue Hospital Medical College, New York.

---

GENTLEMEN,—This is a subject to which I have often referred, but it will bear constant repetition, although many of the points which I shall emphasize may seem quite elementary. Not a week passes in which I do not meet with one or more cases which illustrate the importance of the fact that the general practitioner should be thoroughly instructed as to the etiology and treatment of this symptom; for we must constantly bear in mind that it is a *symptom*, the explanation of which should always be sought for. There is a tendency among the profession to treat every case of menorrhagia expectantly, or, more properly, with drugs, instead of investigating and seeking to remove its cause. There seems to be no reason why the practice in this respect should be so much more superficial than in other branches of medicine. To my mind, nothing is more unscientific than to go on treating menorrhagia month after month with tampons, hot astringent douches, and ergot, allowing the patient to grow weaker and weaker without any attempt to find out why the bleeding persists. This is the mournful history of the majority of inoperable cases of carcinoma. It is not easy to understand how the family physician can be held entirely guiltless under these circumstances.

It is self-evident that in the large majority of cases in which there is a bloody discharge from the vagina, the uterine mucous membrane is the source of the hemorrhage. This may be laid down as a general rule, with but few exceptions. For example, a small intra-uterine polypus not larger than a walnut may give rise to profuse menorrhagia, while in a case of subperitoneal fibroid filling the entire abdomen menstruation may be practically normal.



How shall we account for this difference? Because in one case there is marked hypertrophy of the endometrium, thereby increasing enormously the bleeding surface of the uterus; whereas in the other the uterus is of normal size, and the tumor is so situated that it does not cause that chronic congestion which leads to the development of hyperplastic endometritis. Again, the early hemorrhages of malignant disease, whether it begins in the cervix or in the corpus uteri, are due, not to the neoplasm itself, but to the accompanying endometritis fungosa. In short, in every form of uterine hemorrhage it is our duty to investigate the uterine cavity in the absence of ocular proofs of disease.

No instrument is equal to the examining finger for palpating the endometrium. For some reason tents have fallen into disrepute, and yet it is only when the cervix is thoroughly dilated by means of them that the interior of the uterus can be thoroughly palpated. I have yet to see any harm result from the use of laminaria or tupelo tents, provided that ordinary aseptic precautions are observed and that they are not used in cases of active inflammatory trouble. I need not enter into details regarding the use of tents; suffice it to say that the vagina should be disinfected as thoroughly as for an operation, and that the tents should be sterilized by heat and by immersion in a solution of iodoform and ether just before their introduction. With the exception of puerperal cases, I never examine the uterine cavity except under anæsthesia, and with the understanding that if the cause of the hemorrhage is found I shall be allowed to proceed with the necessary operation, provided that it does not entail the removal of the entire uterus.

With the patient thoroughly anæsthetized and in the lithotomy position, immediately after the removal of the last tent and after thorough disinfection of the vagina, the aseptic finger is inserted into the cervix, and, while the fundus uteri is supported by the external hand, is insinuated into the canal very much the same as it would be worked into the finger of a new glove. With the fingertip every portion of the uterine cavity can be examined, provided that the uterus is movable, is not drawn too far upward by adhesions, or that the case is not complicated by the presence of a large neoplasm. Such an examination positively settles the diagnosis. The condition may be simply hyperplastic endometritis, a small uterine polypus, a sessile fibroid, or localized or diffuse malignant disease of the endometrium.

Curettement naturally follows this procedure, its object being not only to remove diseased tissue, and hence to relieve the symptoms, but to obtain a specimen for microscopical examination. But here let me again emphasize the fact, which is well known but often overlooked, that in cases of sessile fibroid the hemorrhage comes, not from the tumor itself, but from the hypertrophied endometrium. The epithelial covering of the projecting mass is usually atrophied.

Provided that digital examination of the uterine cavity is undesirable or impracticable, there remains the diagnostic use of the curette. Let me caution you against using this instrument in the office. I know that this practice was formerly quite prevalent, and that it is even recommended by some at the present day, but I assure you that it is fraught with no little danger. No instrument should be introduced into the uterine cavity under any circumstances without previous disinfection of the vagina and of the instrument itself. Early in my practice I had a very severe lesson which impressed this on my mind. Slight as the operation is, it is not entirely free from risk. The previous dilatation of the cervix, which is sure to be necessary, has in many instances been attended with laceration of the cervix or perforation of the uterine wall. Only two days ago I was called in haste by a colleague, a very careful man, who, while using a Sims's dilator and exerting only moderate force, ruptured the cervix through the left broad ligament into the peritoneal cavity, tearing the uterine artery. When I reached the scene he was compressing the artery with his finger, the patient being still under an anæsthetic. I succeeded in grasping the vessel with a clamp and arresting the hemorrhage. I then passed three fingers into the peritoneal cavity, assured myself that there was no internal bleeding, and introduced a gauze drain. (The patient recovered without rise of temperature.) This accident has occurred to me twice without any bad results, but operators have not always been so fortunate. A number of cases of cœliotomy for internal hemorrhage, due to rupture of the uterus during dilatation, have been reported, some of which terminated fatally.

Curettement is an operation to be conducted under anæsthesia and with all the usual precautions. It is not necessary to enter into the details of this operation, or to mention in what cases the endometrium should be thoroughly scraped, and when we should be content with the removal of a small portion of the morbid tissue for examination by the pathologist.

It is remarkable that in spite of the widespread intelligence of the profession and the amount of literature on this subject, uterine hemorrhage is still treated expectantly by many excellent practitioners. The routine use of ergot is particularly reprehensible. We forget the elementary fact that ergot really irritates the uterine muscular fibres, that it causes, not normal, regular contractions, but irregular, spasmodic ones, and that so far from controlling hemorrhage, it frequently increases it. It is the common practice to administer the drug in large doses during the menstrual flow, and then to suspend its use after the hemorrhage has ceased. When we reflect that, according to the well-known physiological law, stimulation of the vasomotor nerves causing contraction of blood-vessels is followed by corresponding dilatation, it must be evident that the very agent which causes a lessening of the blood-supply may also produce an increased flow of blood to the parts. It has usually seemed to me more rational, if ergot is used at all, to administer it in small doses during the inter-menstrual period, combined with strychnine and hydrastine or hydrastinine, and to suspend its use during the menstrual flow.

Within the last few days I have seen several cases which illustrate the curious hesitancy which physicians have in seeking for the cause of menorrhagia. A week or two ago I examined under ether and curetted a lady who had had menorrhagia for several months, and during the past two or three weeks had been flowing more or less continuously, so that she had become quite anæmic. From the history it was possible to exclude any inflammatory pelvic trouble, pregnancy, and the ordinary causes of hemorrhage. She had no pain, and it was evident that whatever was wrong, the trouble must be inside of the uterus. A single scrape with the curette brought away fully half a drachm of soft, brain-like material, which even a tyro would have recognized as evidence of malignant tissue. In this case hysterectomy was clearly indicated. A physician's wife had had menorrhagia for months, and yet had never been examined until a short time before I saw her. The vagina was filled with a globular mass, which was recognized as a fibrous tumor. It was easily twisted off, and the patient was cured. A day or two afterwards I saw in consultation a lady, forty-seven years of age, who had had various climacteric symptoms, although she had never entirely ceased to menstruate. She began to have menorrhagia six months before, and

during the past month had flowed almost continuously. Examination showed inoperable carcinoma of the cervix, which extended into the uterine cavity and involved the vaginal vault.

This leads me to touch upon that large class of cases to which I have already alluded, in which menorrhagia appears near the time of the climacteric. Such cases should always be viewed with suspicion, even in the absence of other symptoms. It is time that you should divest yourselves of the notion that pain, cachexia, and a foul discharge are necessarily present in connection with advanced malignant disease of the uterus. I know that even in recent editions of popular text-books this erroneous idea is still fostered. Hemorrhage is the symptom to which you must direct your attention. If you wait for the other evidences of malignant disease to appear, you will usually wait until it has advanced too far for operative treatment. Regard with suspicion every increase of the normal menstrual flow; it may be simply an indication of the approaching menopause, but in any case it is your duty to investigate and to determine positively whether cancer is or is not present. Do not allow any false delicacy on the part of the patient to stand in your way, but insist upon a vaginal examination. It is better to examine twenty women unnecessarily and to be able to assure them that their symptoms are entirely physiological, than to omit this routine practice and thus to overlook a single case of cancer. If you review the histories of the majority of patients in the chronic ward of the Cancer Hospital, you will find that almost without exception they will state that they were assured by their physician that their symptoms were due only to the change of life, and when they were finally examined they were told that it was impossible to do any operation. It is rather difficult for the specialist to explain matters satisfactorily to the friends in some of these cases, because it must be evident, even to the laity, that the hemorrhages had persisted for a long time before any effort was made to ascertain the cause.

Fortunately, we happen to have two new patients to-day who illustrate pretty well some of the points upon which I have been dwelling. The first one whom we shall examine is a girl, eighteen years of age, who has been married two years. She had a child nine months ago. She states that the labor was not particularly difficult, but she was in some hospital for two months afterwards with severe pain in the abdomen, and fever. She can give no clear history of the treatment and does not even remember the hospital, but she was

undoubtedly delivered by some midwife and became septic afterwards. Her symptoms, briefly stated, are: Menstruation recurring at intervals of a little over two weeks and lasting from eight to ten days. The flow is very profuse and is attended with considerable pain in the abdomen. On examination we find a bilateral laceration of the cervix, and extending from the angles of the tears are indurations in the bases of both broad ligaments. The uterus is large and soft, and, though in its normal position, is not movable. No evidence of inflammatory disease of the adnexa is to be found, and no neoplasm can be felt. Now, this is evidently, from the history as well as from the local condition, a case of subinvolution of the uterus with accompanying hyperplastic endometritis; and the indications, according to what I have already said, are to dilate the cervix carefully—this is one of the cases in which you will need to be careful on account of the cicatrices—and to curette thoroughly. I have no doubt that we shall remove a quantity of so-called fungosities. I shall send the patient into the hospital, and you will be able to see the operation later.

The second patient is quite the opposite of the former. She is fifty-eight years of age, and has been married thirty-nine years. She states that she menstruated regularly until fifteen months ago; then she skipped several months, and then flowed profusely. Early in January of this year she began to flow, and the hemorrhage continued for three weeks. She has had severe darting pains in the back and abdomen, and states that her health has declined. She has had some vague climacteric symptoms, such as flushing. This is just one of the cases to which I alluded, in which we may suspect the possible development of malignant disease; but, of course, we should keep this suspicion to ourselves. On exposing the external genitals, the diagnosis is at once apparent. The patient has a complete procidentia, with bilateral laceration of the cervix, without any erosion. By vaginal, also by rectal, examination we find the uterus quite small; the tubes and ovaries cannot be felt. We are then able to state at once that this hemorrhage is probably an ordinary climacteric symptom. Now, how easy and satisfactory is this solution, and why should we not follow this course in every case? The patient will not object to it if it is presented to her in a proper way, and the doctor's mind is greatly relieved.

She states that she has had this prolapsus for a great many years. It does not give rise to marked symptoms, and the question of opera-

tion must be settled entirely by her. Of course any local treatment, such as the use of tampons and astringent injections, would be entirely valueless, as there has been such an extensive laceration of the pelvic floor and over-stretching of the vaginal tissues, that the uterus has absolutely no support. This case would formerly have been treated by plastic operations on the vagina, the usual method being to curette, amputate the cervix, and perform anterior and posterior colporrhaphy. In my experience this is followed by permanent results. I remember in one case that I did four or five plastic operations at one sitting, and two months afterwards it was hardly possible to tell that anything had been done. By performing ventral fixation, supplemented by posterior colporrhaphy, the patient was cured. Alexander's operation would also be inapplicable to such cases as this. Two courses are open: Either to close the vagina by Le Fort's operation, which would hardly be applicable in a case like this, in which the husband is living; or, better, to suspend the uterus after performing abdominal section, and then to repair the lesion of the pelvic floor. I have also employed Freund's purse-string suture in such cases with fair results. The patient will consider about an operation, and if she decides to enter the hospital you will have an opportunity to see it later.

I have, of course, only touched on the causes of uterine hemorrhage. In some cases they are extremely obscure, and are evidently due to general, rather than to local conditions. For example, a lady was under my care a few years ago for obstinate menorrhagia. She had been thoroughly curetted twice without result. Having lived in a malarial country, it was thought that it might be due to malarial poisoning, but the prolonged use of quinine and Warburg's tincture, etc., failed to produce any effect. I noticed that her muscular system was extremely flabby; her uterus was large and soft, and evidently shared in the general muscular want of tone. It occurred to me that if the uterine muscle could be stimulated, this alone, without any further local treatment, would control the flow. I accordingly put her on small doses of ergotine and strychnine during the intermenstrual period, suspending it when the flow began. I gave her twice a week intra-uterine faradization. At the beginning of the period she was placed in the recumbent posture, and the foot of the bed was elevated about eight inches.

At first there was no perceptible improvement. At the second period a marked reduction in the amount of the flow was noted, and,

in brief, this improvement continued until six or eight months afterwards, when the flow was quite normal. In the mean time she had been much improved in her general health by massage, exercise, baths, etc. This is simply an illustration of the fact that hemorrhage may be due to general as well as local causes, and, consequently, in seeking to arrive at the solution of the problem you must not confine your attention entirely to the pelvis, but must investigate other organs, and take a broad, intelligent view of the subject.

There is another form of hemorrhage to which I have not alluded, and that is metrorrhagia, or irregular flow in the inter-menstrual period. In a young woman one thinks naturally of one of two causes—incomplete abortion and ectopic gestation. And here let me emphasize the fact that you must never lose sight of the possibility of extra-uterine pregnancy in every case in which a woman, after passing a menstrual period for a few days, has an irregular flow of blood, whether accompanied by pain and symptoms of pregnancy or not. Suspect that this condition may be present, and then proceed to exclude it by keeping the patient under careful observation and demonstrating the absence of any tumor outside of the uterus.

Metrorrhagia, or an occasional show of blood in a patient who has passed the menopause, is an ominous symptom. I have spoken of that so frequently that such repetition may seem wearisome, and yet I cannot insist too strongly upon the necessity of a local examination in the case of every woman who, having ceased to menstruate for several months or years, has a slight escape of blood following coitus, straining at stool, or any unusual muscular exertion. Examine such a woman without delay, even if this is the only symptom present, because many times it is a precursor of serious trouble, and it is precisely by such opportune examinations that you detect malignant disease in its incipient stage, when a radical operation offers the best prospect of a permanent cure.

I have in this fragmentary way tried to outline a broad subject. The one point upon which I have laid the most stress is *not* to treat expectantly any case of uterine hemorrhage. There is a cause for it, and it is your business to find out the cause. If you fail to do it, if you keep on with palliative means, either hoping that nature will cure the patient, or that the hemorrhages will cease after the change of life, you are simply trusting to luck, and are not acting the part of the intelligent, scientific physician.

# Ophthalmology.

---

## THE RHEUMATIC AND GOUTY DIATHESSES AND THEIR RELATION TO DISEASES OF THE EYE.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL  
SCHOOL AND HOSPITAL.

BY WILLIAM OLIVER MOORE, M.D. (Columbia),  
Professor of Diseases of the Eye and Ear in the New York Post-Graduate Medical  
School and Hospital; Attending Ophthalmic Surgeon to the Orphans'  
Home and Asylum of the Protestant Episcopal Church,  
New York; Visiting Surgeon to the Post-  
Graduate Hospital, etc., New York.

---

GENTLEMEN,—“Forty years didst Thou sustain them in the wilderness, so that they lacked nothing; their clothes waxed not old and their feet swelled not.” We thus read in an old Hebrew book, and in it we have the key to good health and sanitary law. For if we of the present day and generation would live in the wilderness, properly clad, and would eat and drink only such things as are really needed for the continuance of life, we too might say “their feet have not swelled these forty years.”

In the present day gout and rheumatism are observed to prevail wherever there is an upper class having abundant means of self-indulgence, and living without regard to the primeval law of humanity. “In the sweat of thy face shalt thou eat bread.” A well-marked hereditary tendency to these diseases may be observed which even a very active and temperate life may not overcome; while, on the other hand, the grossest forms of excess may be practised for a whole lifetime without incurring a gouty or rheumatic diathesis. It is difficult to explain these variations, but they leave unaffected the general principle that gout is a condition especially of those who have little physical exercise and give great scope to their bodily appetites. With a few special exceptions, those who labor in the



open air are seldom attacked by this disease; those who labor much with the mind and are not restrained by abstemiousness are remarkably subject to gout. Gout and rheumatism are thus the counterpoise in the scales of fortune to many worldly advantages. Hippocrates, in his "Aphorisms," speaks of gout as occurring most frequently in the spring and autumn, and mentions the fact that women are more liable to it than men. Again, Seneca, writing later, mentions the prevalence of gout and rheumatism in Roman women of his day as one of the results of their high living and debauchery. Cullen recognized gout as manifesting itself in various ways,—viz., as regular gout, irregular gout, etc.; and Dr. Garrod adopts a division somewhat similar to, though simpler than, that of Cullen,—i.e., regular gout that affects the joints, and is either acute or chronic, and irregular gout, affecting non-articular tissues. It is this latter form that we have mostly to deal with in affections of the eye; in some cases we see these eye affections in the so-called "suppressed gout." In speaking of these different eye-diseases, which I attribute to rheumatism and gout, I distinctly wish it understood at the outset that I have carefully eliminated any question of syphilis as a cause, knowing full well that many will raise this as a potent factor. How far rheumatism and gout may be related to syphilis I do not know, but I believe that through the process of time the syphilitic and other poisons introduced into the system long years before must become so attenuated and diluted as to produce, in the offspring, tissue changes causing what we to-day term rheumatic and gouty conditions; so that gout and rheumatism may be cousins, so to speak, *far removed*. Their special treatment is essentially different from that of the parent disease.

I shall speak of the ocular diseases produced by rheumatism and gout in an indiscriminate way, believing as I do that they are practically akin, only with different features, as members of one family may differ in external form, one from the other, yet have the same common parentage. I believe that no disease, as such, is inherited, but that dispositions, tastes, and inclinations are, and that offspring inheriting a weak and delicate constitution may have engrafted upon it any of the germ diseases; or, in the struggle for assimilation and appropriation of food elements, have faulty conditions develop, producing one or the other form of systemic disease, such as gout and

rheumatism, but I do not believe that they, as such, are transmissible.

Rheumatism and gout are the result of suboxidation of the proteid substances, either from over-indulgence in animal or vegetable substances, alone or combined. This being a fact, it is more easy to explain why some, following out a certain line of life, have these affections, and others, doing exactly the same, have perfect immunity. In one the oxidation goes on perfectly, and in the other, for instance, the organs that have charge of these processes are at fault, and rheumatism results in some part of the organism. "Thus, by the suboxidation theory, all the pathological changes and symptoms commonly known as pertaining to the various forms of rheumatism are explained." (Porter.)

That these two diseases should attack the eyeball and its appendages at first glance may seem strange, yet when we stop to consider it, it is very clear and reasonable, for we all know that gout and rheumatism primarily attack the joints and prefer the fibrous tissue elements of the body. Do not the eyeball and the orbit in which it is placed practically constitute a joint? The orbit, a bony cavity of considerable size, lined by periosteum, is filled by the eyeball, whose outer covering is in every particular similar to fibrous tissue,—i.e., the sclera and the cornea,—and attached to these coats are muscles and tendinous connections, fulfilling in every particular the requirements of a well-regulated joint. It is not strange, therefore, when viewed in this light, that this organ should be the subject of gouty and rheumatic affections, although we frequently see cases where this fact has been very long overlooked, and the vision damaged in consequence.

I shall first speak of diseases of the orbit and superficial tissues of the eye, and then, finally, of the intraocular tissues that we find involved. The writer of fifty years ago paid much more attention to rheumatism and gout as causes of disease than we; the pendulum swings, and to-day it seems to be towards the germ end of the curve, and with a particular tendency towards syphilis. My endeavor will be, however, only to state the results of practical clinical observation, extending over a considerable period, and to show you the cases; and although I expect to be accused of including the whole domain of ocular diseases within my grasp, I can truly say I have left for future consideration much to be said.

The first case to be presented will be one of *periostitis of the orbit*. This condition is a form of rheumatic or gouty affection, and may be either acute or chronic. It is, next to that of syphilitic origin, most common. The course of the acute affection varies, and the after-results depend upon the promptness with which the inflammatory products are evacuated from the orbit. If early, then the tissues rapidly resume their normal condition; or, if delayed, the pus burrows and general inflammation of the orbit ensues, causing caries and necrosis of the bones, with fistulæ and subsequent retraction of the upper eyelid.

#### ORBITAL PERIOSTITIS.

The patient before you is a male, thirty years of age, with no trace of syphilis, but the subject of rheumatism of the acute variety. When first seen, the right eye was prominent, the lid very much swollen and inflamed, and deep pressure showed pus in the orbital tissue. An incision has been made into the orbit through the upper lid, from which a large amount of pus has escaped. The periosteum is denuded on the inner and upper part of the orbit to the extent of about half an inch (or over one centimetre). The treatment consists in washing out the orbital space with carbolic acid, and giving sodium salicylate internally. This patient will probably require treatment for three months; then we shall expect to find the fistula closed, and the eye occupying its natural position. This prognosis is based upon an experience with a former case. I have only seen two such in my private practice.

The symptoms of these acute cases do not materially differ from other forms of periostitis, the pain being the prominent symptom. In some, the pressure of the fluids contained in the orbit causes inflammation of the sheath, and even of the substance of the optic nerve, causing thereby sudden blindness without any appreciable ophthalmoscopic appearance. Dr. H. D. Noyes has reported a case of rheumatic periostitis in a woman, where the presence of rheumatism was well established. The course of the disease, when chronic, is very slow, lasting months, and, therefore, the ocular troubles do not always develop, owing to the pressure increasing gradually, either from exuded material or exostoses. Occasionally, however, we see exophthalmos and turgidity of the conjunctival blood-vessels. The fistulæ that form lead to denuded bone, and the

skin of the upper lid is retracted, causing ectropion. A point in the differential diagnosis between orbital cellulitis and periostitis is, that in the latter pressure on the margin of the orbit causes great tenderness and pain, whereas in cellulitis this sign is absent. This point was first mentioned by Mr. John Hamilton, of Dublin. When the deep parts only of the orbit are affected by cellulitis, this sign, just mentioned, fails. In orbital periostitis the eyeballs are not so exophthalmic as in cellulitis.

#### TENONITIS.

Tenonitis is another form of rheumatic affection of the eyeball. Santos Fernandez has reported two cases, and Dassart asserts that amblyopia and atrophy of the optic nerve may be of rheumatic origin, these conditions being produced by an exudation within Tenon's capsule, followed by symptoms of compression, slight exophthalmos, amblyopia, and dilatation of the retinal veins. He distinguishes two forms of tenonitis,—one chiefly characterized by chemosis, and the other without it. It is the latter form that is followed by amblyopia and optic nerve atrophy, any chemosis serving as an outlet for morbid secretion in the capsule. The symptoms are those of ordinary cellulitis of the orbit.

I have no case of this to present to you at this time, but I will relate the history of two seen by me, both males, in which no cause other than rheumatism could be given. Both of these patients had previously had acute rheumatism. In one, thirty-two years of age, this swelling of the lids and conjunctiva was marked, and, after evacuation of the pus, the symptoms gradually subsided without any intraocular changes. In the other case, twenty-five years of age, only one eye was affected. Marked swelling of the eyelids took place, but there was no chemotic conjunctiva, although the whole eyeball was deeply injected and the tissues of the eyeball were firm and brawny. This condition continued for two weeks, and then gradually subsided. Ophthalmoscopic examination during the first week revealed well-marked neuritis with considerable swelling. This finally gave way at the end of one month, and six months later there was the appearance of optic nerve atrophy, with vision of 20/L. In the fellow-eye vision was 20/15. The patient, seen two years after the acute inflammation, showed an atrophic optic nerve condition and the same vision as above.

**AFFECTIONS OF THE INTRINSIC AND EXTRINSIC MUSCLES OF THE EYE.**

Paralysis of accommodation has been noticed after severe forms of acute rheumatism and gout. That rheumatism affects the muscles of the eyeball there can be no question, as I have frequently seen patients who complained of lameness on moving the eyeballs, which could not be explained in any other way. During the past two years, especially during the prevalence of "the grip," I have seen a large number of patients convalescent from this disease, whose sole difficulty was this lameness of the extrinsic muscles, giving rise to muscular asthenopia. The administration of sodium salicylate corrects the difficulty. I have also seen the ocular muscles become entirely disabled, the neuritis either arising from the systemic cause, or from pressure on the nerve-trunk from exudations in the orbital space. In such cases we must use both local and constitutional measures,—internally, the sodium and lithium salicylates, and, locally, massage to the affected muscles, with the application of castor- or olive-oil inunctions to the temple and eyeball. Turkish and Russian baths are most excellent in these cases.

**CONJUNCTIVITIS.**

I have no doubt as to rheumatic and gouty conjunctivitis, as three male patients of this class come for treatment several times a year, one of whom I present to you. The conjunctivitis usually antedates the gouty attack. One has learned to take heed of the ocular warning, and the moment the conjunctiva becomes affected he puts himself on his favorite remedy. These cases of conjunctivitis have very little mucous secretion, yet there is some, and the attacks respond quickly to proper internal medication and to local applications to the eyelids of alum, in crystal. Le Roy and others have reported several similar cases. Subconjunctival extravasation of blood has been noticed, and is valuable as indicative of the general systemic condition. Encysted calcareous deposits (urate of sodium) along the upper lid edges are of very frequent occurrence, and are also found filling the Meibomian follicles. So long as they remain in the follicle, beneath the conjunctiva, no difficulty is experienced, but as soon as they ulcerate or protrude through this membrane, inflammation ensues, with lachrymation and photophobia. True pannus may be produced. These little calculi may be picked out

with a delicately pointed instrument, such as a discission needle or a tenotome. All close observers must have noticed them. I have invariably found them in rheumatic persons.

#### ECZEMA OF THE LID.

A scaly eruption of the eyelids is also a sequel of the gouty condition, and this eczema may give much trouble from the attendant itching.

#### KERATITIS.

Rheumatic keratitis is an affection entirely by itself, and limited to its own tissue. It is certainly very rare, although not so rare as some observers would have us believe. These cases run their course without any trace of suppuration, and in the milder forms are manifested by obscuration of the cornea, which looks like a piece of glass when breathed upon. The ciliary injection is very marked, and the ocular conjunctiva is somewhat œdematous, with photophobia, and lachrymation out of proportion to the other symptoms. (The patient is here presented.) The cloudiness of the cornea often changes very rapidly, due to changes in the lymphatic circulation of the cornea. The more common type is where the cornea and iris are both involved, and the iritis in these cases is usually of a serious form.

The case presented has recently been under observation,—a male, thirty-six years of age, who, until constitutional treatment was thought of, remained most obstinate to treatment. The administration of sodium salicylate changed the picture completely. Mr. Jonathan Hutchinson has reported several such cases of keratitis due to rheumatism. In one there was iritis associated with it, and the change from the moist cold of England to the tropics cured the patient. We have seen in gouty persons over forty years of age a form of keratitis characterized by a porcelain-like opacity of the cornea, giving one the impression that the sclera had encroached upon the cornea. A band-like opacity of the cornea is always suspicious of gout. I have seen several cases where the corneal epithelium would desquamate and superficial ulcers form on the corneal surface, first in one, and then in the other eye, causing the patient vast discomfort and pain, and, in fact, keeping him from using his eyes. Here there is no possible question as to the cause. The use of cocaine and castor oil in this case gave great relief.

## SCLERITIS AND EPISCLERITIS.

Scleritis usually occurs in middle life, though it may occur in youth. Its occurrence without external cause, and relapses in various portions of one or both eyes in those carrying out a correct regimen, make it probable that there is a causal connection with some general disturbance of health. De Wecker, Alt, Panas, Zychon, Emmert, of modern writers, and Mackenzie, Laurence, and others, of fifty years ago, refer it to gout and rheumatism. It scarcely ever occurs in a simple or isolated form as a consequence of syphilis. Scleritis attacks only that part of the sclera anterior to the equator of the eyeball, and the inflammation may be either superficial or deep. The superficial form—episcleritis—appears as a circumscribed patch close to the corneal margin, and is often found without pain and with very little, if any, swelling of the parts. It is apt to recur in different parts of the eyeball, leaving slight discoloration of a dusky hue, but no harm comes to the eye. These attacks vary in length of time from a few days to several weeks. In deep scleritis, the inflammation being so violent, thinning of the sclera occurs, and subsequent bulging takes place, presenting a blue-gray appearance, due to the choroidal tissues showing through the thin sclera. Either with this condition, or not, opacity of the cornea is very apt to occur in the course of the disease; it continues and remains as a permanent opacity, causing more or less diminution of vision. The blood-vessels of the sclera become increased in size, and advance towards the corneal circumference, and occasionally over its border. They are bright red, and about equally surround the corneal edge. No secretion, except an increased flow of tears, is noticed, and only very rarely chemosis is found. Dimness of vision is frequently seen, owing to the cloudiness of the aqueous humor. Iritis may be met with occasionally with scleritis, and cases of acute glaucoma have developed. The pain is at first of a stinging kind, from the eyeball to the orbit, and is usually increased by heat. It often extends to the head and temple, and even to the teeth. The eyebrow is, however, its chief seat, and the pain is worse at night. (See patient presented.) There is sometimes general disturbance of the system. In some the attack is very severe; in others only moderate in severity. The subacute form frequently occurs in those who never had rheumatism, but a rheumatic tendency; we rarely

see it as a result of metastasis. Usually it is said by the patient to be due to exposure or refrigeration when the head or face has been very warm. It is more prevalent in the winter and spring when the northeast winds are blowing, and is apt to recur, and is eminently of rheumatic and gouty origin.

The treatment of episcleritis and scleritis is constitutional, by the internal administration of lithium or sodium salicylate, or both; the hypodermic injection of pilocarpine muriate, as advised by De Wecker, and antipyrin, antifebrin, or phenacetin for the relief of the pain. Iodide of potassium may also be found of service. Local applications to the eye of warm fomentations, and the instillation of atropine sulphate, alone or combined with cocaine, in watery solution or in oil, every three hours, will be found to be of great service in allaying pain. Occasional cases are met with where bandaging the eye gives much comfort. Scarifying the inflamed part is commended by Schöler, and the use of the galvano-cautery over the inflamed region is practised by some.

Regulating the quantity and quality of the ingested food is of prime importance. I have frequently seen cases of this affection treated for a long time with only moderate success by local measures alone, where by adding the proper constitutional medication the disease speedily was overcome. In my experience, the salicylates have been most successful.

#### IRITIS.

Rheumatic or gouty iritis is next in frequency to that produced by syphilis, and differs from the other forms only in its obstinateness to local treatment. It has no pathognomonic sign by which it can be known by the ocular symptoms present. The attack is usually sudden, and not infrequently both eyes are simultaneously affected. The symptoms most prominent are contraction of the pupil and impaired mobility, and a general, murky, dull look to the surface, due to exudations into the aqueous chamber, and also to the turgid condition of the blood-vessels. The redness and vascularity incident to these changes are not so pronounced as in some other forms of iritic inflammation. The vascularity is greatest in the ciliary region and corneal circumference, fading gradually towards the conjunctiva. The pain is usually very severe, and extends throughout all the branches of the fifth nerve, more especially the supra- and infra-



orbital branches, and, as in most iritic inflammation, it is worse at night. As a rule, no exudation into the pupil or anterior chamber occurs. Occasionally, another form is noticed in people where, in addition to the symptoms above stated, fibrinous exudates into the anterior chamber take place, so that it is turbid with exudates. This variety is very prone to recur, and is very susceptible to climatic influence. The pain in rheumatic iritis is often very severe, as has been stated, yet often what is called "quiet iritis" is due to the same faulty condition of the system. It has been observed by some German writers that the color of the ciliary zone in this variety of iritis is more dull, and in some cases livid. I am of the opinion, however, that save the peculiar obstinateness of the iritis to the ordinary local methods used in other forms, and the quick response to anti-rheumatic remedies, when given, there are no signs in the eye alone to indicate its cause. The temperament of the individual and the previous occurrence or present existence of rheumatism or gout aid in the diagnosis. The prognosis is good, unless the case does not come under observation early, when posterior synechia form, and, remaining fixed, give rise to subsequent attacks of cyclitis. After an attack of gouty inflammation in the foot we see the parts become tumid and remain sensitive, and the most trivial condition causes a relapse. The same thing occurs in the eye, but here we have the advantage of seeing the vascular changes, and can more rapidly combat any recurrence of the disease. Puch thinks he has seen cases of iritis at the menstrual inauguration that were due to the rheumatic diathesis.

#### TREATMENT OF GOUTY AND RHEUMATIC IRITIS.

Locally, as in iritis of whatever form, the treatment is by mydriatics, as atropine, cocaine, or the two combined in solution. The application of hot water to the closed eyelids, several times a day, is of immense service in relieving the pain. If the pain be not relieved in this way, the hypodermic injection of morphine sulphate, or the use of pilocarpine in the same manner, is beneficial. One of the peculiarities of these forms of iritis is, as stated before, their obstinateness to local treatment, and, therefore, *only when remedies are directed to the constitutional disorder will the ocular trouble disappear*. A valuable way to use atropine sulphate in all forms of iritis, especially where the iris does not completely dilate, is the

rapid method, or, as it is sometimes termed, *coup sur coup*,—i.e., the instilling of the mydriatic every ten minutes for a given hour, twice daily, morning and evening, and in the interval every two hours. Leeches, either natural or artificial, are occasionally needed to be applied to the temple. The general treatment offers great relief. Turkish and Russian baths give great comfort, and with the internal administration of sodium salicylate, either alone or combined with quinine, they hasten the cure. Colchicine, employed in the form of granules (two milligrammes each), increased until the patient experiences intestinal disturbance, has been found excellent by Darrier. In some cases the internal administration of the oil of gaultheria has done better than its fellow, salicylic acid.

#### CHOROIDITIS AND IRIDOCOROIDITIS.

When gout attacks the iris and ciliary body, acute glaucoma is sometimes set up. Boucheron says that the symptoms of gouty or rheumatic cyclitis vary according to the part of the ciliary region involved, leading to supraorbital neuralgia or cephalalgic radiation. To-day, we must all admit that there are many cases of glaucoma directly produced by a gouty or rheumatic condition of the system; the circulation in the blood of the products of suboxidation brings about disturbances in the intraocular circulation, causing pressure and a glaucomatous condition. Noyes has seen a case in which glaucoma occurred during convalescence from acute rheumatism in a young woman twenty years of age, one eye being affected. I have repeatedly seen cases which, to my mind, have had rheumatism or gout as the exciting cause. Glaucoma, as known to us at the present day, was by the old writers ascribed almost altogether to an inflammation which started, as they thought, in the sclera, and progressively deeper and deeper structures were attacked, until finally a general inflammation of the anterior tunics took place, resulting in cataract and hopeless blindness. The increased intraocular tension in glaucoma is due to the distention of the blood-vessels, and associated with this increased tension there is arteriosclerosis and functional disturbance of the heart. Richey lays great stress on the length and course of the *venæ vorticosæ*, saying that these pass in the meshes of the sclerotic downward and backward, the shortest of them measuring one and a half millimetres in length, or only half as long as the sclerotic is thick at its thickest part. With the high

arterial tension found in gout, the arteries become engorged and, as a result, the *arteria centralis* presses upon the vein and causes distention. The moment there is an increase in the intraocular pressure, pressure is made on the *venæ vorticosæ*. This causes venous stasis, which, in turn, increases the intraocular pressure. In this way, he claims, a vicious circle is established.

#### TREATMENT OF CHOROIDITIS AND ACUTE GLAUCOMA.

In simple choroiditis with detachment of the retina, as a result of rheumatism or gout, the hypodermic use of pilocarpine muriate gives the very best result. This treatment, combined with rest, will effect relief. Sodium salicylate may also be given in full doses. In acute glaucoma no especially different plan is to be pursued. Eserine sulphate instilled into the eye every two hours will cut short the attack, if the patient is seen in the first few days. If vision continues to fail, iridectomy should be at once advised. Those having glaucoma that can be traced to either of the conditions of which we are speaking, should be warned of the danger to the unaffected eye, and be put upon proper hygienic treatment in order that they may avoid a similar attack in the fellow-eye. The affection of the horse formerly called "moon blindness," "periodic ophthalmia," etc., is really an iridochoroiditis produced by gout or rheumatism, due to suboxidation of the ingested food; for it occurs usually in young animals who are suddenly taken from pasture and placed in the confinement of a stable, and the food changed from grass to grain.

#### OPTIC NEURITIS.

It is the well-formed opinion of the closest clinical observers that there is no doubt whatever that the rheumatic and gouty diatheses are the cause occasionally of optic neuritis. I have seen two such cases in the past ten years. The one presented to your notice is a male, twenty-nine years of age, the neuritis being binocular and associated with a swelling of 3 D in the right eye and 4 D in the left eye. Vision was 20/30 in each eye when first seen. The patient had acute rheumatism eight months before in the knee-joint, and recovered after a severe illness of six months. Another attack of only a week's duration in one knee antedated the ocular symptoms by two weeks. No syphilis or other cause could be discovered. The blood and urine were carefully examined, with negative re-

sults except as to rheumatism. The case has improved as to the neuritis, while vision has failed in each eye to 20/C, with the appearance of optic nerve atrophy.

The other case is a woman, thirty-two years of age, who had binocular optic neuritis following a sharp attack of acute rheumatism in each shoulder. Her hands also showed the well-known deformed knuckles. The nerve-head was swollen to 3 D. After six weeks the neuritis subsided, and finally, eight months later, the nerves looked pale and atrophic, and vision was 20/70 in each eye. The local affection receives no peculiar character from the constitutional disposition, nor does it require any peculiar local treatment. Hutchinson reports a case of double optic neuritis in a patient where there was no discernible cause except an inherited tendency to gout; and also one of monocular neuritis with vitreous opacities in a lad of sixteen. Fulton has also reported a similar monocular case of neuritis due to rheumatism.

The treatment should be directed especially to the general cause, and the antirheumatic and gouty remedies pushed vigorously and aided by Turkish and Russian baths. Locally, leeches should be applied to the temple. Many have expressed great doubt as to either rheumatism or gout producing double or monocular neuroretinitis, but we cannot understand why they object, for certainly a deposit, either gouty or rheumatic in character, can exist inside of the skull as well as on its outer surface. If so, why not these conditions produced by such deposits? Such deposits in the skull pressing on the origin of the optic nerves, or on their continuity, would produce either double or one-sided neuritis, the same as any other exudation.

#### RETINITIS HEMORRHAGICA.

We have on several occasions seen retinitis with hemorrhages due to gout, where the general condition had not produced any cardiac or renal disease. Both of these conditions have been carefully looked for. The only possible factor in the production of the retinal hemorrhage seemed to be the atheromatous retinal vessels. I have two such cases now under treatment, one of whom is shown you,—a man, forty-nine years of age, who has had, and still suffers from, gout in the feet. The failure of vision took place quite suddenly in the right eye, and when first seen the vision was 20/50. The ophthalmoscope showed a small hemorrhage to the outer side

of the nerve and below the macula lutea. The vision is now 20/30 and the clot is almost absorbed. Careful constitutional treatment has been insisted upon, and no local treatment was adopted.

The other case, a male, forty-eight years of age, is also the subject of gout in the feet. His history is almost a duplicate of the first, except that the left eye was the one affected. Vision was 20/70 when first seen. It has been gradually improving until it is now 20/30. The hemorrhages have been absorbed. In this case it had encroached nearly upon the macula. It is interesting to note that in each of these cases the ocular symptoms came on in the interval of the attack, when the patient was in good health(?). No renal or cardiac disease was apparent. In all these cases of retinal hemorrhages from gout or rheumatism the condition is supposed to be due to a retinal arterio-phlebo-sclerosis, and they are usually found in the early stages of the disease. Another important symptom has been the finding of yellowish granular exudations in the retina, generally leaving the macula intact until late in the disease. Changes in the optic nerve are generally intraocular. Occasionally deposits of uric acid between the choroid and retina are found.

#### OPACITIES IN THE VITREOUS.

Many cases of opacities in the vitreous are found, supposed to be due to the faulty metabolism, producing gout or rheumatism, where hemorrhages are found in the vitreous, and by some authorities they are looked upon as pathognomonic of gout. The changes in the vitreous are usually bilateral, though rarely symmetrical. They have been frequently found to relapse, clearing up as the uric acid has diminished from the urine, and coming on again with its increase in the system. Hirschberg has witnessed a crystalline deposit in the neighborhood of the macula in such a case, and another observer has found the blood-vessels of the fundus obliterated by deposits of urates. Treatment of the constitutional disorder is all that is required in these cases.

#### CATARACT.

It has been said, with good reason, that when the physiological hardening of the lens is associated with loss of transparency, there is good ground for believing that there is an underlying pathological condition, and in the opinion of many the gouty and rheumatic

diatheses are the common cause of chronic and subacute inflammation of the uveal tract. The entire choroidal tract will be found more or less involved in every case, and in some diabetics certain changes in refraction are apparently due to changes in density of the fluid brought about by alterations in the composition of the blood. If this is true of diabetics, it is not improbable that similar changes in the nutrition of the lens may result from alterations occurring in the blood in connection with a gouty or rheumatic diathesis. It is a common observation that in patients presenting themselves with cataract a history of gout or rheumatism will be universally admitted, and that the alleviation of the diathesis will make rapid changes in the appearance of the lens. As an instance, I will relate to you the case of a patient upon whom a cataract extraction was done, both lenses being opaque, and the patient being decidedly gouty. The health of the patient was deteriorated, not only from the gout and from the loss of vision, but also from the fact that she was very much depressed by her condition, being a woman of business and fearing the loss of her occupation. The successful extraction of one cataract so far improved her physical and mental condition that the other cataract after three months entirely disappeared. This I ascribed entirely to the improvement in her physical condition. The patient having one good eye, and being sixty-five years of age, learned to ride a bicycle, and now takes much more exercise than formerly. The increased exercise and the improvement in her mental condition have brought about an improvement in her gouty condition, hence the transparency of the lens, formerly opaque. Many cases of so-called cataract are simply temporary changes in the lens, due to uric acid in the blood, causing striæ to appear, and on the blood again becoming normal the striæ disappear. Hence, gentlemen, we see the necessity of keeping the condition of the blood pure.

**OPERATION FOR THE EXTRACTION OF CATARACT  
AND FOR SECONDARY CATARACT; ASTHEN-  
OPIA FROM HYPERMETROPIA; PATHOLOGY OF  
SYMPATHETIC EYE-DISEASES; SYPHILITIC IRI-  
TIS.**

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY THOMAS R. POOLEY, M.D.,

Professor of Ophthalmology in the New York Polyclinic; Surgeon-in-Chief to the  
New Amsterdam Eye and Ear Hospital, New York City, New York.

---

GENTLEMEN,—I present to you a patient, seventy-six years of age, who nearly three years ago had his left eye subjected to the operation for cataract by my distinguished colleague, Dr. Knapp. The operation was that known as “simple extraction,” which is but a return to an old method, in which it is again essayed to remove the cataract without making an iridectomy. The method is as old as the history of cataract itself. After the method of depressing the lens into the vitreous, and leaving it there, this operation was the one which was adopted, excepting that the method for extrusion of the cataract was more distinctly a flap operation, that it lay more in the cornea, and that it was made with a Beers knife. This method was practised exclusively for many years, both in Europe and in this country. It was known as Beers’s operation for flap extraction. It was almost entirely abandoned upon the introduction of Graefe’s method. Graefe attempted an entirely different plan of operation. He began and finished his section, not in the cornea, but in the sclera, and a beautifully planned Graefe’s section made in this way left nothing to be desired, so far as coaptation of the wound was concerned. Of course, after the section, iridectomy was performed. Very soon objections to this method arose. The wound, when made in the sclera and planned exactly after the classical operation of Graefe, sometimes gave rise to iridocyclitis, and many eyes were lost. Again, in the hands of operators not so skilful as von Graefe, the

vitreous was lost, or the wound was placed too far in the sclera, and inflammation very promptly followed the operation. This led, not to the abandonment of Graefe's operation, but to its modification, and we find Graefe modifying his own operation. This modification consisted in placing the wound distinctly within the corneal margin, although still adhering to a section which was rather a linear wound than a flap. I cannot go into detail, but I may say that after many years of practice of Graefe's modified linear extraction, which became the generally received operation, we have a return to extraction without iridectomy, but very much the same kind of a wound is retained. At present, in contradistinction to many of my colleagues, I am still in favor of Graefe's extraction in the majority of cases, and I have many reasons for it; and one of them, which is to me the most conclusive, is that I am perfectly sure of what I can do with Graefe's extraction. I approach extraction of cataract with almost positive certainty of being successful if I employ Graefe's method. If I do simple extraction, I must learn a new surgical technique, and I may have to do this at the cost of my patient. There can be no doubt that where simple extraction has been accomplished without accident, and there are no complications, the result is as beautiful as can be obtained in any surgical procedure. You have extracted the lens, you have left a perfectly circular iris, and a central and movable pupil,—in fact, there is nothing more to be desired. In Graefe's operation, on the contrary, no matter how successful the extraction, it is at the expense of a coloboma, or a gap in the iris, which certainly has its drawbacks, for sight cannot be so perfect through a large immovable pupil, which must of necessity always remain wide, and in which vision is accomplished under circles of dispersion.

Let us now look at the other side of the question. We are told by the partisans of the operation that the objections which I will bring forward do not occur. I say that one of the greatest objections to simple extraction is the fact that you so often see prolapse of the iris. One of the greatest dangers for the subsequent safety of the eye, after a surgical procedure of any kind, is the same that obtains after an injury if the iris remain engaged in the wound. The advocates of the operation say that only about five per cent. of the cases have such a prolapse, but this represents a number of cases in which the eye is menaced. This, it is true, is a pretty low percentage. Admitting this percentage, upon that showing, even, I



think the operation more dangerous than Graefe's, but I am not converted to the opinion or belief that either three or five per cent. begins to cover the number of cases in which prolapse of the iris occurs after simple extraction. It may more than cover the cases in which it occurs during the operative procedure, but there are cases where this accident takes place after the patients have passed from observation. The foregoing remarks have led me to present to you this gentleman.

He was operated upon nearly three years ago. The night after the operation he had intense pain in and about the eye. He remained in the hospital a little over two weeks after the operation, and the healing was quite favorable. This gentleman now comes to me for advice about his eye, and I will ask you to examine him with me. If I lift his lid so that you can see the wound, you will find exactly in the centre of the scar a small incarceration of the iris. When you illuminate his eye with oblique light, or with the ophthalmoscope, you see a dense capsular membrane which fills the entire pupillary area, and as he looks upward, you will observe that not only is the iris drawn up towards the apex of the wound, and is adherent there, but the capsule of the lens follows the same direction. We have then a small incarcerated hernia of the iris and of the capsule. Fortunately, in this case, it is not likely to be of moment, as, after the lapse of three years, he has as good vision as one could expect through the dense capsular membrane which remains. I propose to divide this secondary cataract. At present, with plus eight diopters, he has vision only of  $\frac{20}{100}$ , and this is of but little practical value, as it only enables one to distinguish large objects. I do not hesitate in such cases to make a discission of the secondary membrane, unless the acuteness of vision is all that one can reasonably expect. In cases where the capsular membrane is at fault, you will frequently triple or quadruple the vision by such an operation. As I illuminate the eye you can even see the manner in which the capsulotomy was performed. In the centre of this capsular membrane there seems to be a triangular membrane. The capsular membrane is not so dense in its centre, which makes it look as though there were a small pupil there. You cannot expect to obtain perfect vision through such a pupil. If you make traction on the capsular membrane, you invite an inflammatory reaction, and hence the method of tearing with needles I consider very improper. Looking at this

patient's right eye you will see there is a decidedly opaque reflex from the centre of the lens, due to beginning sclerosis of the centre or nucleus of the lens,—in other words, a beginning nuclear cataract. Examining the periphery of the lens, we see striæ of opacities, and if examined with the ophthalmoscope, they can be found both in the anterior and posterior sectors of the lens. We have then opacities both in the centre and in the anterior and posterior cortices of his lens,—a senile cataract, developing in the usual way. There is a perfectly good reflex from the background of the eye, so that it will be a long time before the cataract becomes mature.

In regard to the operation for discission, I think in every case a cutting instrument should be employed; an instrument like a needle will never suffice for these operations. An instrument is required which has a minute blade shaped like a crescent, with a cutting edge on one side, and so constructed that when the knife is entered through the cornea into the anterior chamber its shaft exactly fills the opening, and prevents the escape of the aqueous humor, for a retention of this humor as long as possible is an absolute necessity for the nice performance of the operation. An obstacle to the use of the sickle-shaped knife is that you must have excellent illumination and operate with great care, or else you may find yourself cutting with the back of the knife; hence, I prefer the spear-shaped knife with a double cutting edge. You should never operate without a dilated pupil, and, above all, without cocaine, for cocaine not only gives you the advantage of anæsthesia of the cornea, but the very great advantage that, if the eye be thoroughly cocainized, the escape of the aqueous humor is very much retarded, owing to the loss of tone of the eye produced by the cocaine. With the pupil dilated, the instrument is to be introduced into the cornea a little to the temporal side of the pupil, and just a little below the vertical meridian of the eyeball. Many of my colleagues operate without fixing the eye, but, although this way looks very brilliant, it is in reality bad technique, and you cannot secure the same nicety of manipulation. If you introduce the instrument too obliquely, you will not enter the chamber at all. The instrument is carried over to the nasal side, and then, the cutting edge being turned directly backward, a horizontal incision along the lower part of the pupil is made. This should never be a tearing procedure. Having laid open the desired area by an incision, the handle of the instrument is moved

around until its upper part is vertical, and you reach nearly the upper margin of the pupil, when you make another cut in a similar way, which will meet the first one at either an obtuse or a right angle. This operation must always be done by oblique illumination, the patient being in a darkened room, and so situated that the assistant can throw the light directly into the pupil with a large lens of long focal length. After the operation, the eye is bandaged, and, if there be no reaction, the patient will be well in from four days to a week.

There is a procedure known as iridotomy, to which I wish to allude. Let us suppose that instead of being merely a capsular membrane, this membrane is also partly a product of inflammation,—that there has been an iritis following an operation, and much inflammatory material, which glues the capsule and the iris together. The capsular membrane is resilient, and when it is cut, retracts, but not so with this inflammatory exudate, and hence, the best way to operate in these cases is one in which an opening is made through this dense diaphragm, and the sphincter edge of the iris is divided. To do this, introduce a Beers knife at the lower sclero-corneal margin in such a way as to perforate the whole dense diaphragm towards the vitreous humor, and at the same time cut the sphincter edge of the iris. Take an instrument known as Tyrrel's hook, and introducing it into the opening made, engage the hook in the mass, and slowly draw it into the lips of the wound, and cut it off. In this way you will obtain a pupil with less injury to the eye than by any other method I have seen described. This method I learned from Dr. Knapp. In regard to the operations performed in secondary membranes, I may say that they should be performed early, as after awhile the membrane becomes tough.

This young woman, twenty-seven years of age, comes to us complaining of pain over the eyebrows, which has been pretty constant for the last two months. This headache is present on rising in the morning, and is worse after using the eyes. She came to me the other day, and the examination showed that there was hypermetropia. If you will inquire into the history of a great many cases of headache you will often find that they depend upon errors of refraction, and at the very head of this list I would put hypermetropia. This patient has been under medicinal treatment for some time without benefit. Her right eye has vision  $\frac{2}{10}$ , and this has

been found to be rather better with a convex glass. In the other glass the vision is  $\frac{30}{80}$ , and she sees just as well with plus one diopter. We have, then, established the diagnosis of hypermetropia in both eyes. Examining this patient with the ophthalmoscope, we find that the amount of hypermetropia is a great deal more than that indicated by the examination with the test glasses. The fundus could be seen with a very much stronger convex glass than she accepts to see with in the distance. If we are perfectly sure of our own and of the patient's accommodation being relaxed, we can determine with the ophthalmoscope exactly the amount of the error of refraction; but, unfortunately, both these conditions are rarely present at once. Hence, it is far better to paralyze the accommodation, using either sulphate of atropine or homatropine. If the element of time is of no importance, you may use a one-per-cent. solution of sulphate of atropine, instilled three or four times a day, for several days, and by this method a perfect paralysis of accommodation can be secured; but as most people cannot spare this time, homatropine is very desirable, for the paralysis which it produces does not last so long. This patient was directed to instil a solution of homatropine this morning at intervals of fifteen minutes for an hour. The effects will pass off sufficiently within twenty-four hours to allow the use of the eyes. We shall now find that her vision in the distance will not be so good, and she will accept a much stronger glass. If you examine a patient without atropine, and you find he sees just as well or better with a convex glass, you say there is hypermetropia. You must remember that all young hypermetropes accommodate even for the distance at which you examine them, and they will give you very hasty and inaccurate answers if you are not on your guard. You must try each glass slowly and patiently before deciding upon what glass is best suited to correct the error of refraction. The strongest convex glass with which a patient can see at a distance is the equivalent of the manifest hypermetropia,—i.e., the amount of hypermetropia found when the patient is accommodating. There is also latent hypermetropia. If the latent hypermetropia be brought out by the use of atropine, then you have the absolute hypermetropia, and it is a very nice question in the examination of these patients to decide as to whether atropine should be employed or not. Ordinarily, for people who are well advanced in years towards middle life, I am willing to examine and correct

the manifest hypermetropia, giving the patients the strongest convex glass with which they can see in the distance; but in young subjects, and in people who show with the ophthalmoscope a considerable amount of latent hypermetropia, I always use atropine. If you find astigmatism, the use of atropine is still more imperative.

We have not time in this lecture to carry out the subsequent steps of the examination, but on examining her eyes now with convex glasses, it will be found that she will take a much stronger glass than before the use of the homatropine. Another examination will be made after the effect of the homatropine has passed away. The rule is to prescribe a glass which represents the difference between the latent and the manifest hypermetropia; but I am sure that, in many cases of asthenopia, we never give the patient perfect comfort until we give a glass which represents the measure of the absolute hypermetropia.

#### THE PATHOLOGY OF SYMPATHETIC EYE DISEASES.

We now come to speak of the pathology of sympathetic eye diseases. We find ourselves at once in a domain where there is much dispute as to the variety to forms of expression of sympathetic disease. There is no doubt at all that the commonest manifestation of sympathetic eye trouble is iridocyclitis, and this too may be said to be the most dangerous. It will certainly comprise a majority of the cases which come under our care. All of the several tunics, however, according to authorities, may be sympathetically affected. Thus authors describe sympathetic keratitis, scleritis, and conjunctivitis. I think we may disregard any affection of the cornea as of sympathetic origin. It does not seem to me, even from the statement of those who have described these cases, that they come under the head of what we are studying,—a disease transmitted from one eye to the other.

I will first consider, then, the varieties of sympathetic affections of the iris and ciliary body. The most innocuous and least to be dreaded of the forms of sympathetic disease is serous iritis. This manifests itself subjectively by faint circumcorneal injection, by a more or less intolerance to light, very little pain, and impairment of sight. From the very insidious and almost painless approach of serous iritis it is generally quite far advanced before it is seen by a physician.

If you examine the eye sympathetically affected in this way,

you will find that the pupil is not contracted, but often the reverse, and the anterior chamber seems to have increased in depth. The iris is not discolored, there is no exudation in the area of the pupil, nor is there synechia; but on examination by a weak light you see deposits of lymph or plastic material on the posterior layer of the cornea. This affection has been variously described as *keratitis punctata*, *iritis serosa*, etc.; but I think the proper term is *iritis serosa*,—i.e., one in which there is a tendency to a serous exudation, which is deposited on the posterior layer of the cornea. This is the least dangerous form of the disease. I cannot agree with Mauthner as to this form of sympathetic eye-disease being so generally cured, for there is great danger of its developing into iritis or iridocyclitis. Under favorable auspices the eye is not irretrievably lost, as it is in *iritis plastica*.

If we now take up the consideration of sympathetic iridocyclitis we notice first that it is an exceedingly grave affection. It declares itself from within ten days to a number of years after the eye has, as a result of injury or disease, become affected with iridocyclitis. It, too, becomes frequently affected in a very insidious manner, so that the sight is often hopelessly compromised before being seen by a physician. Owing to the painless onset and progress of sympathetic disease, it is a wise precaution to examine the condition of the sound eye at every visit. In *iritis plastica* there is a tendency to formation of plastic material which comes from the ciliary bodies, and is exuded into the posterior chamber, where it forms broad synechia iris to the capsule of the lens. There is also an exudation which fills or occludes the pupil. The broad synechia which touch the surface of the iris even extend entirely around the pupillary opening in the iris, and then we have a condition known as exclusion of the pupil. Now the exudation occurs between this shut-off portion of the iris and the more peripheral parts in the posterior chamber, and the iris becomes distended by the exudation behind it, so that it presents a very peculiar appearance. There are often little balloon-like elevations in the iris, which correspond to the points of the iris which are held by the synechia, the part of the iris remaining distensible being distended by the exudation. Another peculiar appearance of the iris is the retraction of the ciliary part of the iris, and at the same time the advancement of the pupillary portion of the iris towards the posterior surface of the cornea forming the

"crater-shaped pupil." This exudation now fills the pupil so that the pupil is not only excluded, but occluded. Now the natural intercommunication between the anterior and posterior chambers is shut off, and the result is that we have in some of these cases during the course of iridocyclitis a veritable expression of glaucoma, the eye becoming hard. This exudation, however, is not likely to last very long. It is only one phase of the development of cyclitis. As the disease progresses more and more, the iris becomes vascular, and you see a swollen, sodden, and vascular condition of its structure, and as this condition in turn gives rise to atrophy of the iris tissue, there is in the late phases of iridocyclitis a general loss of nutrition of the eye, the wasting of the globe, known as phthisis bulbi. During the course of the disease, which lasts for weeks, months, and even years, this process continues.

During all this time the condition of the eye may vary. We have simply the fact that there is iridocyclitis in this eye, perhaps chronic in character, showing itself by the ciliary regions still remaining painful and by occurrence of attacks of inflammation of a painful character in the lost eye, or, if the disease be more recent, we have almost the identical phases of the disease that we have in the sympathetically affected one.

If we consider now the sympathetic affections in one of the tunics of the eye, I think we must admit beyond doubt that there is a sympathetic inflammation of the optic nerve and retina. I was among the first to describe the fact that during the course of sympathetic iridocyclitis an inflammation of the retina and optic nerve occurs. I reported in the early numbers of Knapp's *Archives* two cases of sympathetic ophthalmia, characterized by the occurrence of neuroretinitis, and I reported two cases in which, during the course of iridocyclitis, inflammation of the optic nerve and retina was observed. I found upon subsequent observation that Graefe had, however, described and published the same thing, but my article was among the earliest publications of the kind in this country.

If you observe neuroretinitis under these circumstances, it is almost impossible to show that the inflammation has not extended through by the extending of the inflammation from before backward, affecting in turn the choroid, the retina, and optic nerve, hence the cases of this kind are not pure neuroretinitis of a sympathetic character.

Shortly after this publication other cases were described. I published not long afterwards one or two cases in which there were sympathetic neuroretinitis without any inflammation of the uveal tract.

I will describe briefly my own observations. A patient came to me whose eye had been injured by a blow from a twig of a tree. The eye was lost as a result of this injury. When I saw him I found the lost eye was painful; although there was no ciliary injection or evidence of active inflammation in the other eye, there was some dimness of vision. An ophthalmoscopic examination showed a most marked neuroretinitis, the disk margin being obscured by exudation, the pupillary area swollen, and the vessels hyperæmic. I showed these cases to Dr. Knapp, who concurred in the diagnosis. The injured eye was removed and the patient kept in bed in a darkened room, with the result of securing rapid improvement, and permanent improvement of the sympathetically affected eye. While these cases are, to my mind, indisputable signs of a sympathetic disease, they do not form an argument against the transmission of disease through the ciliary nerves. It is likely that in cases of pure neuroretinitis, where there is no inflammation of the iris or ciliary bodies, that the transmission is through the optic chiasm. In short, I do not think it is logical for us to assume that every case of sympathetic inflammation is caused in the same way.

Neuroretinitis may, then, occur as a phase of inflammation which attacks the whole uveal tract and may appear as a primary affection.

Whether we can have inflammations of other parts of the eye which are strictly due to sympathetic disease, I do not think profitable to discuss here at present, although it might be profitable in an extensive treatise on this subject. The cases often described as sympathetic glaucoma are really instances of sympathetic iridocyclitis, and as a result of the exclusion, or occlusion, of the pupil, there is an increase of tension of the eyeball. But this is only an incident in the course of iridocyclitis. It must be borne in mind, of course, that in transmitted inflammatory diseases one may meet a great variety of symptoms, but we are chiefly concerned with the pathological conditions, which result in inflammatory troubles, transmitted through the injured or lost eye to the sound one, and I think in all of these cases we must say that we have only to consider inflammations of the uveal tract or inflammations of the optic nerve or retina.

The patient showed you the last time is still improving. The



circumcorneal injection is disappearing and the point is entirely gone. As soon as the cyclitis had lost its painful character there was no longer any danger.

#### SYPHILITIC IRITIS.

This man presents a very interesting case from a clinical standpoint. The diagnosis is iritis. He came to me six days ago and admitted having contracted syphilis twenty-eight years ago. In speaking of iritis, I told you that the commonest cause of this condition was syphilis, and that it was most apt to occur in the early stages of secondary diseases, although there are exceptions to this rule. This attack of iritis began about five weeks ago, and in an insidious manner, and this is very common in syphilitic iritis. Before coming under my care he had been treated several weeks by another physician. At the time I first saw him there was a synechia downward and inward, which was not even broken so late as yesterday, although to-day even this has yielded and the pupil is completely dilated. One week of vigorous treatment with atropine, aided by the application of leeches and the administration of mercury, has brought about this gratifying improvement; while during the four weeks previous to this, although the treatment followed the same general lines, dilatation of the pupil had not been effected, and this I attribute to the fact that most general practitioners do not use strong enough solutions of atropine, nor is it employed vigorously enough. If you wish to break up synechia, the drug must be used very energetically, and the instillations must be made every hour or two. It is very common to see cases in which at first the atropine only produces a very insignificant dilatation of the pupils, in which by frequent instillations and the applications of leeches these adhesions are broken up. In an old synechia case like this, I think we should employ mixed treatment, or iodide of potassium alone, but in the earliest stages I would probably make use of mercurial inunctions. Examining this patient with oblique light, you notice the pigment on the lens showing where the synechia have been touched. The atropine will be continued so long as there is any circumcorneal injection.

# Laryngology and Rhinology.

---

## PARALYSIS OF THE MOTOR OCULI NERVE; ACUTE CATARRHAL INFLAMMATION OF THE EUSTACHIAN TUBE AND MIDDLE EAR.

CLINICAL LECTURE DELIVERED AT THE HOSPITAL COLLEGE OF MEDICINE.

BY SAMUEL G. DABNEY, M.D.,

Professor of Physiology and Clinical Lecturer on Diseases of the Eye, Ear, Nose, and Throat in the Hospital College of Medicine, etc., Louisville, Kentucky.

---

GENTLEMEN,—This patient, Mrs. J., is a widow, fifty-eight years old. You will observe that there is a constant trembling of the head and limbs. It is easily seen when I ask her to hold her hand out in this way. She tells us that this condition has been present for a great number of years without especial change for better or worse. It is called paralysis agitans. She comes to us, however, for a disease manifested in the eye, though having its real seat in the nervous system. Our neurologist, Dr. Moren, is of the opinion that there is no connection between the paralysis agitans and this ocular disturbance, so we will treat of the latter without reference to the former condition. The patient tells us that some weeks ago she suddenly observed that she saw things double, and that she was unable to raise the right upper lid as she did the left one, and, further, that she could not see close objects clearly with the right eye. There has been no pain and no general disturbance except a tendency to vertigo, which is clearly due to the double sight, as it disappears when one eye is closed.

Now, examining her eyes, we notice there is a drooping of the right upper eyelid. The lid is not swollen, and there are no signs of inflammation, and yet she cannot elevate it. In the second place, we observe that the pupil of this eye is semidilated, and that it does not contract on throwing a light into the eye. In the third place,

we observe that the eye is turned somewhat downward and outward, though she is still able to move it to a considerable extent. Finally, we find that she cannot see close objects as clearly with this eye as she can with the other one.

This group of symptoms establishes the diagnosis of a partial paralysis of the motor oculi nerve. You will remember that this nerve supplies (1) the muscle that lifts the upper lid, known as the levator palpebræ superioris. Hence this paralysis accounts for drooping of the lid, which is called ptosis. (2) It supplies the sphincter muscle of the pupil, and so paralysis would account for the dilated pupil which does not respond to light, which is observed here. (3) It supplies the muscle of accommodation, and the loss of this power explains our patient's inability to see close objects distinctly. (4) It supplies all the external muscles of the eye except the external rectus, which turns the eye outward, and the superior oblique, which turns the eye downward and outward, and therefore, when it is paralyzed, the eye, being left to the unbalanced action of these two muscles, is turned downward and outward, as we observe in the case before us.

The paralysis of this nerve, however, is not complete, as is proven by the fact that the lid only partially droops over the eye, and that the eyeball can be moved, though incompletely, in different directions.

We must next try to ascertain where the seat of this paralysis is. You will remember that the motor oculi nerve has its origin in a series of nuclei which lie in the channel that leads from the third to the fourth ventricle of the brain, and which is known as the aqueduct of Sylvius. The nerve, after uniting its fibres from these nuclei, emerges from the base of the brain, passes through the sphenoidal fissure, and goes to its distribution in the structures contained in the orbit. The lesion of its paralysis may therefore be situated either in the nuclei of origin, or along the course of the nerve, or, finally, it might be peripheral. This last location, however, may be excluded in this case with great certainty, because so many different muscles are involved. We are to determine, therefore, whether the disease is nuclear in its origin—that is, situated in the aqueduct of Sylvius—or whether it is caused by some disorder along the course of the nerve. This question is often difficult to decide. Generally speaking, a partial paralysis involving only certain fibres of the nerve is more apt to be nuclear in origin. More-

over, we can find no evidence of pressure in the orbit or along the course of the motor oculi, and, finally, the suddenness of its onset is also indicative of nuclear disease.

Our diagnosis, then, is partial paralysis of the motor oculi nerve, due to a lesion or lesions in the aqueduct of Sylvius.

Now as to the etiology of this affection. Except for the paralysis agitating our patient's history is entirely negative. The ophthalmoscopic condition is normal. There are no evidences of brain tumor or of inflammation. She describes no symptoms indicating specific disease. It is well, in this connection, as Dr. Noyes says, to remember the rule in whist, "When in doubt, play trumps." So in such affections as this, when in doubt, we give iodide of potassium and mercury. It is not fair to conclude that improvement under these means is proof positive of a specific disease, and yet the presumptive evidence is strongly in favor of it.

I tell you now that this patient has been under this line of treatment for the last week, and has very decidedly improved. So the cause may fairly be attributed to syphilis.

The treatment, of course, in all such cases must depend upon the etiology.

The prognosis varies. In the case before us, it is rather favorable, as specific treatment has already produced great improvement, but you must always remember that syphilis of the nervous system is a far different thing from the same disease elsewhere. It frequently produces here changes of structure and loss of function which no amount of treatment can restore.

Among the ocular symptoms that often indicate disease of the nervous system two are of especial importance to the general practitioner, because they may be easily distinguished without the use of the ophthalmoscope or any special testing of the vision. These symptoms are a dilated pupil, which does not respond to light, and a paralysis of one or more of the external ocular muscles manifested by double sight. This double sight may last from a few minutes to an indefinite length of time. It is often transient, and returns from time to time. Either of these symptoms may exist alone, or the mydriasis and diplopia may be combined. In either event, the condition is often the precursor of a serious disease of the nervous system.

Frequently such symptoms precede locomotor ataxia. Many years may elapse before distinct evidences of this affection are mani-

fested, or it may rapidly ensue after an interval of only a few months. As illustrating the importance of this, let me relate the following case: Some years ago a gentleman of this city, a lawyer by profession, applied to an insurance company for a policy on his life. The examiner found him a favorable risk, except for a dilated and irresponsive pupil on one side. On this account he was referred to me for examination. Except for the mydriasis and partial loss of accommodation there were no symptoms of ocular disturbance. These conditions were evidently central in origin, and because of them a policy was refused him. A few months later he applied to the examiner of another insurance company, who either overlooked or disregarded the pupillary condition. The man received a policy for \$10,000, and a few months later symptoms of locomotor ataxia rapidly increased, and within a few months the man was dead. Instances more or less similar to this are by no means rare. Remember, however, that the interval is frequently a long one before the manifestation of other symptoms.

Finally, we see cases of unilateral mydriasis occasionally which are never followed by any subsequent disease of the nervous system. In a recent text-book on diseases of the eye the gripe is mentioned as an occasional cause of a dilated pupil, and I have seen one case in which no other cause could be found.

In general, however, you must remember that most of these cases are of syphilitic origin.

CASE II.—The second case I wish to present to you to-day is this little girl, twelve years of age. We are told that several weeks ago she had an attack of sore throat, followed by certain symptoms in her right ear. These symptoms were ringing in the ears, decided pain, deafness, a stopped-up, muffled feeling, and a peculiar sound of her own voice. The pain in the ear has subsided, and the hearing has somewhat improved, but there is still decided deafness and ringing. Examination of her nose and throat shows a slight catarrhal condition, but no marked obstruction, and no enlargement of the faucial and pharyngeal tonsils.

It is important to examine these structures carefully, because a very large proportion of the acute inflammations of the ear in children are due to adenoid growths.

I will now examine the drum membrane. Holding this round aural speculum by its rim, between my left forefinger and thumb, I insert it into the auditory canal. At the same time, with my right

hand, I pull the auricle upward, backward, and outward, so as to straighten the canal, and I reflect the light from my head-mirror into the ear. It is impossible for you to understand pathological conditions without first having a knowledge of the normal appearances. I recommend you, therefore, to provide yourselves with a head-mirror and an aural speculum, and to examine the healthy ears of some of your friends. Normally the drum membrane should appear grayish-white in color, translucent, sloping from above downward and inward, and presenting on its surface several objects of interest. Of these I will mention only two. They are the handle of the malleus and the light spot. The former is that process of the malleus which is inserted into the membrana tympani, and the latter is due to the reflection of light from a small portion of this membrane. The light spot is usually triangular in shape, and situated a little below and in front of the end of the handle of the malleus.

In the ear of this patient I find that the handle of the malleus appears foreshortened; that the light spot is small, and that the drum membrane is rather lustreless and reddish in color. These changes indicate an inflammation of the middle ear and a retraction of the drum membrane. This last condition is due to the fact that the Eustachian tube is also inflamed. Indeed, it forms the very beginning of the disease, and, as it becomes swollen, the passage of air through it to the middle ear is obstructed. Consequently, the atmospheric pressure bearing on the outer surface of the drum membrane pushes it in, as it is no longer counterbalanced by a similar pressure from the inside. This obstruction to ventilation through the Eustachian tube not only interferes with the hearing, but in many cases causes considerable nervous depression. The patient declares that he feels as if there were a weight bearing on him, and his voice sounds to him as if he were talking in a barrel. The pain is chiefly due to an extension of the inflammation into the middle ear. This disease is called acute catarrhal inflammation of the Eustachian tube and middle ear. Its cause is usually, as you have seen in this case, an acute sore throat or naso-pharyngitis. Its symptoms are well presented in the patient before us.

The prognosis is favorable, if proper treatment is carried out, but if treatment is neglected these cases too often form the beginning of an insidious chronic aural catarrh.

The treatment is as follows: With this atomizer I will spray a mild alkaline and antiseptic solution into this child's nose, and have

her hawk it through her throat. If there was abundant secretion or great relaxation in the naso-pharynx, I would apply, on a curved probe, a solution of iodine and glycerin or nitrate of silver (from ten to thirty grains to the ounce) to this area. Finally, and of chief importance, I blow her ears up with this Politzer bag. This treatment should be repeated daily, or every other day, until the condition is greatly improved, and then at longer intervals.

Other measures are often called for. If seen in the early stage of the disease, when the pain is severe, a douche of hot water is often valuable. Various medicines are recommended, as ear-drops, to allay the pain. I believe that heat is more apt to be efficient than any of them, yet I often use a solution of atropine, morphine, carbolic acid, and cocaine, in the proportion respectively of one-fourth grain, one grain, two drops, and five grains to two drachms of water; five or six drops of this solution should be warmed and put in the ear. Occasionally leeches are needed, though less often than in the suppurative variety of the disease.

When there is an accumulation within the drum membrane, its puncture may be necessary. Internal treatment is often called for to allay the pain and support the health.

**MASTOID CRIES WITHOUT EXTERNAL SIGNS;  
MASTOID SYMPTOMS FROM FURUNCLE; CRIES  
OF THE CANAL SIMULATING FURUNCLE, AND  
TYPICAL MASTOID ABSCESS.**

CLINICAL LECTURE DELIVERED AT THE UNIVERSITY HOSPITAL.

BY B. ALEXANDER RANDALL, M.D.,

Clinical Professor of Ear Disease, University of Pennsylvania; Professor of Otology,  
Philadelphia Polyclinic; Eye and Ear Surgeon to the Methodist and  
Children's Hospitals, etc.

---

GENTLEMEN,—The four cases which I bring before you are instructive in their relation to diagnosis first, and then as to methods of treatment. Each has deceptive elements and might mislead you into a false diagnosis and wrong treatment, with possibly grave results. In the first we have now a complaint of earache of a few days' duration accompanied by signs of slight tympanic inflammation; but as we study his congested drumhead a depressed scar can be noted in its lower back quadrant telling of a past suppuration, while a glance at his mastoid, which is slightly painful and tender to pressure above, shows a depressed scar over the tip of the process. This recalls his condition when I first saw him in this lecture-room five years ago, and pointed out the probable necessity for the operation of which this scar speaks. He came to us suffering with severe pain of this right side and profuse suppuration of this ear after the grippe; and when I saw him was little relieved by the Wilde's incision down upon the mastoid, which Dr. Brown had done because of the pain and tenderness at the mastoid tip. There had been little swelling or redness, and no pus was found; but he gained relief from the bloodletting, perhaps. Really his typical mastoid inflammation symptoms were less than we may usually expect to find in such acute tympanic suppurations. One point was notable, however. Below the mastoid tip there was a distinct, soft swelling, pressing out the sterno-cleido-mastoid and limiting the opening of the mouth by its



tenderness at the angle of the jaw. Fluctuation in this was doubtful and pressure failed to force any pus from it up into the tympanum and increase the flow from his ear; so we waited two days. Then as both these signs were found, we opened over the mastoid tip, found an ounce or more of pus in the digastric fossa and burrowing down along the great blood-vessels, and tracing it back, found the bone sinus on the median aspect of the mastoid. Trephining its thick exterior plate, we cleared the process of much foul pus, flabby granulations, and carious bone, and secured good irrigation through the antrum and out through the drumhead, also down the Eustachian tube, as witnessed his gagging at the irrigations and the purging which followed use of bichloride solution. The healing was kind and fairly prompt, and has remained complete with good hearing until this fresh cold-taking, five years later. This was one of the less usual "digastric" cases, often called by Bezold's name, where mastoid pus breaks inward and burrows down the neck, because less able to penetrate the specially firm exterior of the mastoid than to break through the very thin median aspect. Located in the tip of the process, as many of the grippe empyemas are, it yet communicated rather freely with the tympanum; but if allowed to pursue its course it would have emptied into the mediastinum or pericardium before it would have successfully drained through the auditory meatus, the outer surfaces of the mastoid, or the neck. Hence the great importance of recognizing these cases, however obscure.

This second man presents the mastoid symptoms which the first lacked. You see at a glance the displaced auricle, pushed out and down and forward by the mastoid swelling behind it; and inspection of the auditory canal shows it to be closed by swelling of the upper back wall. This might indicate disease and even empyema of the antrum or be distended by pus burrowing out from the attic: yet cleansing not only shows us a furuncular abscess of the affected region from which the pus can be pressed out, and within which no bone can be felt by the probe, but we are also able to carry a speculum past the stenosis and to see the virtually normal drum-head beyond. As the tuning-fork and other tests of the hearing join in demonstrating the normality of the tympanum, and nothing in history or findings is discrepant, we may assume that we have here to do with a mere furuncle. One point is noteworthy, however: the furuncle, wherever occurring, is usually an affection of the sebaceous

glands, and accordingly superficial. A rarer form is also met, however, involving the sweat-glands or their homologues, the ceruminous glands of the meatus, as my friend Dr. A. Van Harlingen brought to my notice. As these extend their coils into the subcutaneous tissue, we have naturally a deeper lesion in such cases, and in the auditory meatus are apt to see decided involvement of the periosteum, with displacement of the auricle by its cedematous swelling. Such inflammation may go on to caries of the bone surface or even extend into the mastoid cells; but this is probably less frequent in fact than in appearance,—outward extension of disease from the mastoid being mistaken for furuncle.

In most of the furuncular cases, if we cleanse as thoroughly as possible with such an agent as hydrogen dioxide, rub in well a salve of hydrargyri oxidum flavum three to five per cent., and tampon firmly with cotton covered with this, we will have the satisfaction of seeing prompt resolution. The conical tampon, which is put in as tight at first as the patient can bear it, may soon be pressed deeper by the patient with little pain, and serves to reduce hyperæmia, promote absorption of infiltration, and with each jaw motion to massage and rub in the yellow oxide ointment. Only a few cases do not yield to this method. Some patients will not tolerate it, others cannot, the latter being those with periosteal inflammation. In these a free incision to the bone is needed to give due bloodletting and escape of exudation, then the hot douches at short intervals will advance the cure, and the yellow oxide tampon will soon be in place to complete it. This we employ in the present case.

The third case much resembles the second, and may be an instance of either of the three unfavorable alternatives cited with reference to it. Little displacement of the auricle can be detected, although there is œdema about it, except that this ear is lower than the other, as is often natural. The upper wall of the canal is swollen down, closing the lumen; and as we gently employ a speculum as a dilator (one of the few instances where it is justifiable so to do) we reach the mouth of a sinus above, from which pus is being squeezed by its pressure. Within this sinus the probe penetrates deeply, bringing out epithelial and cheesy detritus, flabby granulations bleed easily or are broken off, while some bare bone outlines a considerable loss of substance, of which the soft roof may be granulating *dura mater*. Beyond the sinus we can see only the sagging

upper wall of the meatus and a glimpse of injected drum-head, which becomes moist after cleansing, in evidence that there is flow of pus at the bottom of the canal as well as from the upper wall. Probably we have here one of the cases of attic suppuration with burrowing of pus out along the upper wall of the canal, but more probably with empyema of the mastoid cells above the canal breaking downward into it. Pressure of the probe upon the roof meets some firmness of resistance and causes no faintness or dizziness, so it is not likely that the dura is uncovered; and we will cleanse carefully by probes, curette and syringe, and pack with a shred of gauze, hoping without undue loss of time or protraction of danger to obtain cure without more radical intervention.

This fourth case is said to have had discharge from his right ear for six weeks, but has been ailing and with some ear-pain since the whooping-cough, six months back. He too has an ear displaced by a circumscribed swelling on his mastoid, pressing the auricle forward, out, and down, while amid the sanious pus filling his canal we see a dark polyp reaching almost to the exit. The mastoid swelling is not very tender to pressure, distinctly fluctuates, and in a white child would show some redness, as well as the local heat which you can recognize,—the classic signs of inflammation distinguishing it from a cyst, such as sometimes presents almost identical appearances at this point. This is a typical pus collection upon the mastoid. Etherizing the boy, and shaving and disinfecting the aural region, we will probe the canal before incising, as we were not before able to do. There is bare bone at the posterior margin of the drum-head, and inspissated as well as fluid pus; and the flabby polyp brushes off and comes away whole. Making an incision a little behind the insertion of the auricle and extending from the mastoid tip up nearly three inches we lay bare the pale, thickened periosteum, and then cutting through it give vent to a half ounce of rather cheesy pus. Tuberculosis is so common in the mulatto that we might have expected this; but it is rather more typical than I have often seen it. Laying bare the bone backward for half an inch and forward to the canal, at the upper back edge of which the well-marked *spina supra meatum* is our best landmark, we stanch the moderate bleeding by nipping a few points and sponging firmly, and can then search the surface for a sinus or other evidence of disease. None is manifest; only the rough surface on which the pus lay looks pin-

stuck, yet without the "shaven-beard aspect" often presented. Some men would therefore say they had done enough, and that it would be meddlesome to attack the bone, since the pus may have formed upon its outer surface only. Not once in ten times is this right, and you see that my thumb-nail can crumble the rotten cortex. The chisel and spoon easily open the mastoid and evacuate another half ounce of cheesy pus, friable bone, and unhealthy granulations. Curetting freely in the safe directions, cautiously upward towards the middle fossa, backward towards the sinus, and inward upon the facial and semicircular canals, we soon have a cavity larger than we might have thought his small mastoid could contain. Packing with gauze strips we can thoroughly cleanse it of blood and *débris*, and see that we are scraping pale, rather unhealthy, but now smooth bone, and have not anywhere laid bare the dura. The antrum has been freely opened, and irrigation passes out of the canal; the surfaces are clean and firm and slightly oozing everywhere; the posterior meatus wall, which we bare by pressing the lining of the meatus forward, is bare but not clearly carious; so we put a stitch at the angles of the needlessly large wound and pack with iodoform gauze both the bone cavity and the canal. Perhaps we ought to have removed the ossicles, but my probing did not show them to be bare or carious, and by the history the condition is rather acute and therefore less in need of that step. That the process is tubercular we can have little doubt, and it should therefore be very radically dealt with; since not a few cases begin in such a way but may be checked here or by the excision of affected glands, and saved from further dissemination of the tuberculosis.

Reviewing the cases presented, you will note that no one symptom is decisive in the diagnosis of the various conditions confronting us in this region, even when they are uncombined. Many possibilities must be borne in mind, and excluded one by one through careful investigation, if we are not to rest upon mere guess-work. Experience can make it easier to leap to a conclusion and to explore with a rapidity that looks very superficial, but only by conscientious study can we avoid mistakes in diagnosis leading to sins of omission or commission in treatment, often most serious to our patients.

# Dermatology.

---

## A CASE OF EPITHELIOMA RECURRENS.

CLINICAL LECTURE DELIVERED AT THE ATLANTA MEDICAL COLLEGE.

BY M. B. HUTCHINS, M.D.,

Clinical Lecturer on Dermatology and Syphilology, Atlanta Medical College,  
Atlanta, Georgia.

---

GENTLEMEN,—This case is of especial interest as showing how long an epithelioma may remain superficial, the many different methods of treatment employed, and the remarkable repetitions of returns at the borders of or in the cicatrix. The case is further interesting in that it developed when the patient was only twenty-eight years old, and apparently originated in a sebaceous gland or its duct. It is of interest also because it demonstrates the possibility of multiple centres of development, or that early, superficial metastasis was a marked feature.

I am going to give you the notes as written, almost an exact copy from my case record. Mrs. L. has been a private patient of mine for over three years. Six years before I saw her in January, 1894, there appeared near the root of the nose a pinhead-sized papule, which, she says, became vesicular, then pustular. It failed to heal, but gradually grew. (Before this she had a "white spot" there.) Caustics were applied and salves were used by a New Orleans physician, but were unsuccessful, the disease returning at the border of the scar.

At the time of my examination the whole right side of the bridge of the nose, to the inner canthus of the right eye, was involved. The growth was flatly papulated, firm, waxy in color and appearance, the borders sharply defined, its surface showing many dilated vessels. Here and there the lesion was pitted or excoriated. Just below the inner end of the right brow was a roundish, umbilicated, firm, waxy (semitransparent) lesion about one-half inch in diameter. Both

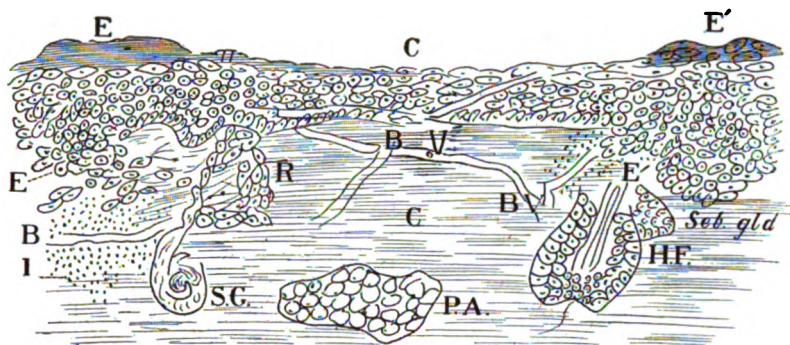


FIG. 1.—The pathology of the recurrence of cancer. *C*, old cicatrix; *E*, points of recurrence; *H F*, hair-follicle; *P A*, panniculus adiposus.

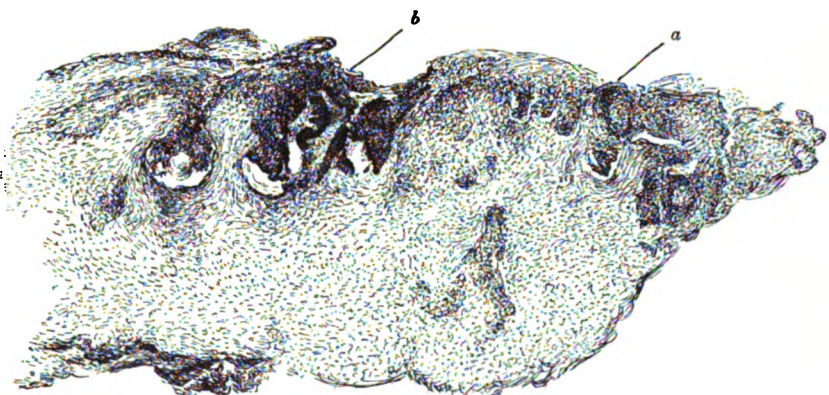


FIG. 2.—Section through the lower eyelid under low amplification. At *a* and *b* horny epithelial ingrowths are shown, in one of which a cavity has formed.

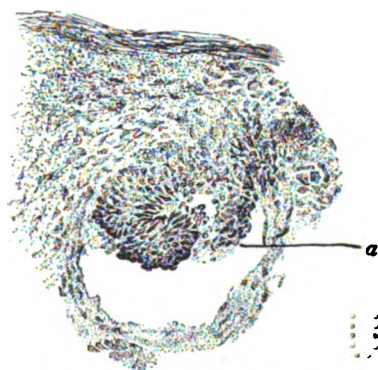


FIG. 3.—More highly magnified mass of cells which are somewhat spindle-shaped towards the centre (*a*).

১৭৬৬

growths were movable with the skin. There was no pain, and the general health was good. The patient was of French extraction.

On January 30, 1894, the two lesions were excised in one piece, under ether anæsthesia and with antiseptic precautions. Antiseptic dressings were used. Four days after the operation the wound was clean and granulating nicely. The dressings thereafter were such as the indications required,—the use of non-aseptic rubber tissue (or carelessness) having permitted infection. The nitrate of silver stick was used to keep down unhealthy granulations.

An attempt at grafting failed, three weeks later.

A month after the operation the wound had healed by granulation, but there was a "suspicious" papule in the upper lid.

About six weeks later it was noted that there were a few large vessels in the scar, a papular growth at the lower side, and ulceration of the papule at the inner end of the upper lid.

Exactly six months after the operation the lid lesion had extended to the inner canthus. A four-per-cent. solution of cocaine was injected and the lesion destroyed with the red-hot, smaller Paquelin point. The eyeball and lids were protected with wet cotton and adhesive plaster. It healed in three weeks. The naso-facial lesion persisted, and there was evidence of remaining disease at the upper edge of the last-treated point.

A little over six months later the lid lesion was clipped out with scissors under a one-per-cent. cocaine solution. It was well in eleven days. The microscope showed the characteristic epitheliomatous structure of the reticular variety. At this time the naso-facial lesion was of bean-size, firm, grayish, thinly scaly.

A month later this was removed under a four-per-cent. cocaine solution by elliptical incisions well around, above, and below the growth. Three stitches were put in. The "skin" part of the incisions united by first intention, the part in the old cicatrix healed by granulation. You can see the line to-day, gentlemen, which shows perfect removal of all disease, and instructs us by the success obtained as to the proper method of excision,—viz., wide of the apparent margins of the disease. On August 30, 1895, a firm, sharply defined infiltration of half-dime size was noted just beneath the inner end of the right brow. The edges were sharply defined in the loose skin, the centre showing a thin, dry crust, chiefly composed of epithelial cells. This lesion was a pure "local recurrence." A line



was made with ink around its margins, and the patient given the ointment of the ammoniate of mercury to apply.

About five months later the lesion was, apparently, destroyed with caustic potash. Two months later a recurrent papule on the lower edge of the nasal scar was similarly treated. This point remains well, as you can see.

On July 3, 1896, another lesion of bean size was destroyed (with caustic potash) at the edge of the scar over the root of the nose.

On March 1, 1897, I pronounced the patient cured. You see how incorrect my opinion was, as we have at least two suspicious lesions to deal with to-day.

I will state here, gentlemen, that the first operation took in part of the upper lid at the inner canthus. Notice it carefully, and see if, now, you can tell that this lid has been touched. The tear-duct is doing its work all right. Now, gentlemen, I want you to look at this, to see how complete and perfect the last excision was. There was a new growth of the disease; I made an excision, and it is completely cured. We must remove all of the disease,—that is the essential point. The present eyelid lesion is exactly where the other one was, right in the scar. There is also one on the nose, at its base on the right side, which we can destroy completely.

It has been claimed that the inflammation around points you cauterize destroys any outlying cells. I used the caustic potash and produced a great deal of inflammation, yet the disease has returned. It is said that inflammation thus aids a cure: I do not know,—experience alone will show whether it harms or whether it does good. The caustic potash did not entirely destroy the cells, for there is a slight recurrence at the root of the nose, right where I used caustic potash last July. I would like you to look at this. Here is the old scar. It is thinner at the sides and less livid than the remainder of the scar.

Now, an epithelioma may develop in one of two or three different ways. Normal epithelium rests upon the basement membrane. Whenever these cells break through the membrane and begin to grow where they formerly did not exist, it is malignant disease. I have taken it for granted that what occurred in this case was that when the first operation was performed a few cells were left here and there in the skin. These multiplied and formed new cells. If two or three cells are left outside the line of operation, they will grow just like the original. This is illustrated by the drawing I have

FIG. 4.—Epithelial down-growths into the corium, with the formation of pearly bodies so characteristic of epitheliomata.



3700

made. There is another way of development which is not so well shown in this drawing,—that is when the epithelioma originates from a tube of a sweat-gland instead of from the epidermis, and forms a net-work beneath the surface of the skin.

#### PATHOLOGY OF RECURRENCE.

I have made a rough sketch of what probably has taken place in this case, gentlemen. (Fig. 1.)

In the middle of the drawing the cicatrix from the former treatment is shown. At the top of the drawing on each side of the cicatrix is represented the so-called recurrences (*E*). The upper part of the whole drawing represents the epidermal part of the skin. At the elevation on the right I have supposed that a part of the disease was left at the edge. On the left the edge of the cicatrix shows normal epidermis, but at the base of the epidermis, on the left of the scar, we presume that some "cancer cells" remained after the treatment. On the right these remains proliferate upward, outward, and downward, forming a new nodule of disease. On the left our few cells became separated and some of them drifted along the lymph-spaces, leaving gaps between themselves and the mother group, to form the nodule at *E*. At *R* is represented a reticulated growth of the cells, which is frequently seen in the epitheliomata called tubular. These latter frequently originate from the sweat-glands or their ducts. A specimen formerly taken from this case showed the reticular form decidedly. Now at the "*B. Vs.*" in the drawing we have the cutaneous vessels, around some of which are seen numerous leucocytes indicating inflammation, this inflammation being due to the irritant action of the epitheliomatous growth. *S. G.* shows a sweat-gland, in the reticular part of the true skin, its canal passing upward to the epidermis. *P. A.* represents a fat lobule constituting a part of the panniculus adiposus. *H. F.* shows a hair-follicle with a sebaceous gland at its neck.

To return to the epitheliomata. The cells multiply, and thus enlarge the primary growth, and some of them become separated and lodge at points away from the original focus,—constituting metastasis, one of the marked features of malignancy. Metastasis occurs late in many epitheliomata, the disease remaining practically localized for a long time. There has never been metastasis to the preaural or other glands in this case. The tubular form is very slow to undergo metastasis.

The central cells of an epitheliomatous nodule become flattened and resemble the most superficial of the epidermal cells, and then, from the centre of the nodule to the edge, they follow in type, to some extent, the cells in order from the surface to the base of the epidermis. Sooner or later the pressure of the new growth cuts off nutrition and death of the tissues begins. Infection adds its injury and we have "a cancer."

#### OPERATION.

We prepare the surface for treatment, inject (an alleged) one-per-cent. solution of cocaine along the lines of intended incision. The nodules at the base of the nose and in the right upper eyelid are excised. The cocaine anæsthesia is very unsatisfactory, and the patient's struggles interfere greatly with the performance of proper excision. Having accidentally cut through diseased tissue I change for another knife, fearing that I might implant "cancer cells" in healthy tissue with the first. The growths are kept for histological study.

The eyelid wound is perfectly closed with silk sutures; the edges of the part at the base of the nose are not coaptated.

Later: Union of the lid wound took place by first intention, the healing of the rest by granulation, all healed rapidly.

Later: Now we come to the reason why excision so often fails in these cases. *We cut too near the visible edge of the growth.* The microscope has shown me that I have done this in the present case, and the patient is not cured. We must cut wide of the disease regardless of the patient's feelings, and the cosmetic result must be wholly a secondary consideration. A confession of error carries more instruction with it than a boast of success. Despite the patient's struggles and the possibility of deformity to the lid I should have made a wider incision.

Fortunately the disease is superficial, with but slight tendency to diffusion, and I consider the case yet within the limits of curability. The proper application of the caustic potash stick will probably complete the cure.

There are two ways to decide whether our excision has included all of the growth. One, the usual, is to await future developments. The other, and more scientific, is to make a histological examination of the part excised. Now the eyelid appears to be entirely cured, but, in spite of this appearance, the very first section made of the edge of the part removed shows the presence of epitheliomatous



**FIG. 5.—Epithelial mass of round, oval, and spindle cells.**



**FIG. 6.—Characteristic appearance of an epithelioma.**

330

structure. This being true, we know that epithelioma remains in the tissues external to the line of excision. The study of specimens from such cases would soon convince any surgeon of the necessity for wider lines of excision.

HISTOLOGY OF THE PARTS EXCISED. (ALCOHOL HARDENING, HÆMATOXYLIN, AND EOSIN STAIN.)

(Fig. 2.) Edge of lid growth. Leitz Oc. 4, Obj. 4 (low power). The first section from the edge of the excised eyelid growth shows an ill-formed epidermis, with no interpapillary cones, but many embryonic ingrowths destined to form hair-follicles or sweat-glands. One of these ingrowths has become keratized. (Fig. 1, *a*.) Near the centre of the epidermal part there is a cavity (Fig. 1, *b*), projecting from the base of which into the corium is an irregular mass of cells, rather lobulated, for the most part showing no basement membrane. They are stained deeply and, save in the centre of the lobules, uniformly. The outermost cells (with Obj. 7, high power) are mostly oval, the central ones are spindle-shaped, and in the centre of some of the larger collections horny. (Fig. 3, *a*.)

Under the low power the dense corium shows but traces of vessels or inflammation. There are many agglomerations of cells arranged for the most part in tubular form, some of these latter being cut longitudinally, others transversely. The high power shows them to be composed mostly of spindle-shaped epithelial cells with a bordering basement membrane. The cells have shrunk away from the basement membrane in many places, and are altogether amorphous in their arrangement. (This specimen is rather thick and not clearly stained.)

Some of the cell groups (the smaller) resemble portions of sweat-canals, others, the larger, hair-follicles, and some even nearly normal sebaceous glands, and this section bears much similarity to irregularly developing embryonal skin.

The next section shows, under the low power, an epidermal structure much like the first, but in many points its lowermost cells have grown, unlimited, down into the corium, as is characteristic of epithelioma. The budding downward of cells is not so marked in this specimen, and the tubular formations are not only present throughout the corium, but they are often directly continuous with the epidermal cells. The tubular sections and the collections resembling hair-follicles and sebaceous glands in the corium give a picture which



bears some resemblance to a section of the normal kidney. Branching of the tubular formations is marked. Evidences of inflammation are present in a somewhat greater degree than in the first section (the section is better prepared). But the inflammatory action is of the slightest. One horny (aborted hair structure) ingrowth is visible. Under the high power the larger cell-collections are still decidedly like hair-follicles and sebaceous glands. A basement membrane is quite constantly present, and there is no tendency of these structures to release metastatic cells.

Section No. 3 is very thin and well stained. (Fig. 4.) It shows a well-defined epidermis. In places the epidermal covering is broken off or portions have fallen out, but here and there cellular down-growths are noticeable, and at one point a pearly body, or nest, surrounded by cells with weakly stained nuclei. (Fig. 4, *a*.) In the corium are many of the tubular formations, near the thinned surface, lying in cavities much too large for them. (Fig. 4, *b*.) Deeper we find a collection of tubular sections partly reticulated, partly isolated. (Fig. 4, *c*, *d*.) In the reticular part of the corium—in places—we find many of the aggregations of cells, resembling hair-follicles and sebaceous glands in the other sections, but here more like epitheliomatous lobules. (Fig. 4, *e*.) There is some—slight—inflammation about the blood-vessels, as shown by leucocytes, and a few connective-tissue cells. (Fig. 4, *f*.)

Under the high power, one of the tubular formations seems to end at a depression of the horny layer (Fig. 4, *g*, and Fig. 5), and is composed of a mass of round and oval cells. The reticulated collection of tubular formations is composed of round, oval, and spindle cells, and portions of it resemble the normal sweat-gland, while other portions seem simply an arrangement of longitudinally placed spindle cells. (Fig. 6.)

Section fourth (of the eyelid specimen) is well prepared. Under the low power we find the epidermis quite well developed, but showing few interpapillary processes. Epitheliomatous down-growth is but faintly present, and such growth is probably but a part of a rudimentary hair-follicle, save in one or two points at the edge of cavities left by the falling out of cells in the preparation of the specimen. In these latter we have clumps of round and spindle cells irregularly arranged and rather closely packed.

The tubular, adenomatous structure is striking in this section.

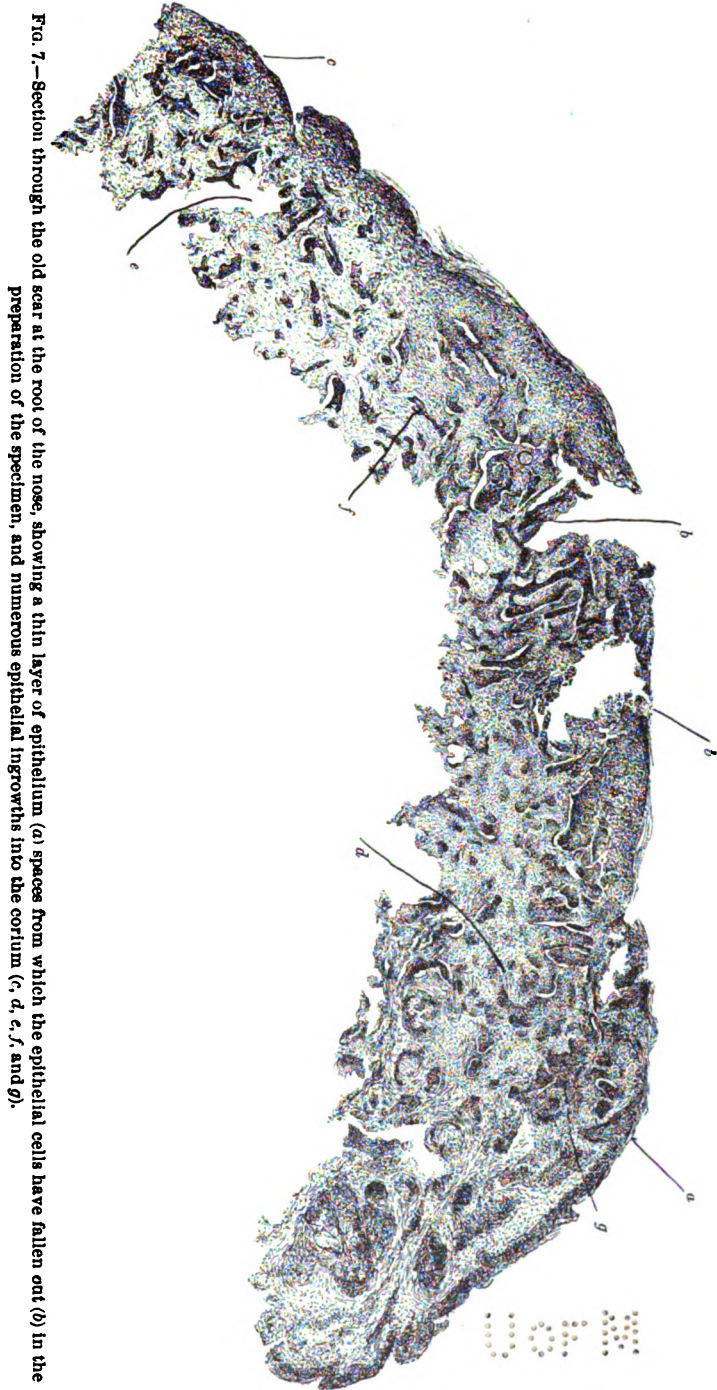


FIG. 7.—Section through the old scar at the root of the nose, showing a thin layer of epithelium (a) spaces from which the epithelial cells have fallen out (b) in the preparation of the specimen, and numerous epithelial ingrowths into the corium (c, d, e, f, and g).

३१७३

Throughout the corium are numerous sections of the straight and branched tubular formations, being specially numerous in the papillary layer, and cavities from which others have fallen. Quite a number of rudimentary hair-follicles, one or two containing a fine, longitudinally cut hair, are present. *This is of marked interest, because of the constantly repeated statement that no hairs occur on the surface of the eyelids.* Histologists, however, describe and figure these fine hairs as normally present. A few sebaceous gland-formations appear deep in the corium. Inflammatory action is but faint and blood-vessels are few. Where the tubular formations predominate the fibres of the corium are densely woven, forming a firm capsule around each of them. The branching of an apparently well-formed sweat-canal is marked in one place in the papillary layer. Under the high power the branched, or single, tubular structures show, where well developed, the formation of the ordinary sweat-glands or canals, the branching one, which lies near the epidermis, sending a canal towards the base of the skin and giving the picture of an inverted sweat-gland and canal. Other branched growths are composed of more or less atypically arranged spindle and round cells. The capsules have a dense fibrous structure.

Here and there are normally formed rudimentary or developed hair-follicles, while there are a number of these structures which have gone awry. Many rounded clumps resemble the Malpighian bodies of the kidney. The epithelial down-growth seen under the low power appears—under the high—to be composed of badly stained, mostly spindle-shaped cells, without a definite limiting membrane.

The structures which resemble sebaceous glands under the low power appear to have undergone a degeneration, gelatinous or fatty in character.

(Section 4 of the eyelid is not represented. It is much like the section of the lesion at the root of the nose, as is shown in Fig. 7.)

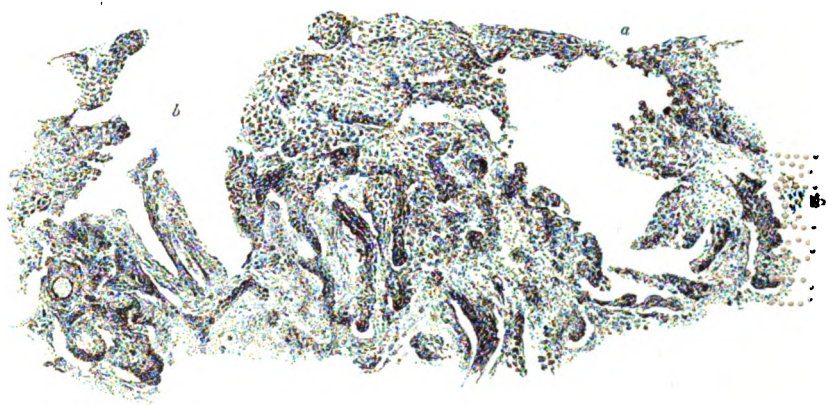
An examination and comparison of the growth which was removed from the edge of the old scar at the root of the nose is of interest. A small part of the section shows, under the low power, a thin epidermis with a few interpapillary cones, and rudimentary follicles and hairs. (Fig. 7, a.) The remainder of the epidermal covering has undergone epitheliomatous change, and we here find the typical disease, a complex of lobular and tubular epithelioma.

In one place a mass due to epithelial proliferation has fallen out of the section (in preparation), and there is a ragged cavity showing projecting masses of cells, save at the base, where a few broken bundles of connective tissue appear. In this region the tubular formations and small hair-follicles are continuous with the epidermal down-growths,—the “lobulated” epithelioma. One follicle projects below. Its upper part is broken at the cavity. (Fig. 7, *b, b'.*)

On the left the early stage of the disease is well marked by the projection downward of processes resembling deformed interpapillary processes. (Fig. 7, *c.*) The corium shows marked inflammation, and numerous tubular, rounded, and sebaceous gland-like growths (Fig. 7, *d, e*), while infiltration of the free spaces with epithelial cells is distinct. (Fig. 7, *f.*) Rudimentary hairs and their sebaceous glands are present here and there. The deepest part of the section shows practically normal connective tissue. Even below the normal epidermis, in the upper reticular part of the corium, are numerous infiltrating epithelial cells. (Fig. 7, *g.*) Under the high power the masses in the edges of a small cavity are found to be composed of amorphously arranged round and oval cells. The horny epidermic cells on each side of the cavity's edge have shared the general hypertrophy, elsewhere it is thinned. The masses around the large cavity are simply conglomerate cells growing atypically. The cells stained poorly. (Fig. 8, *a, b.*)

To sum up the result of the histological examination, we have a compound epithelioma. There is difficulty in deciding which structure furnished the starting point of the disease. We have seen budding downward of cell-growths, to form structures resembling sweat-canals and glands and hair-follicles. In other sections there has appeared proliferation of the epidermal cells in the manner usual in lobulated epithelioma. Whether all the epithelial structures of the skin were affected simultaneously or in turn we are unable to say. It is possible that the adenomatous formations appeared first, as shown by the budding processes, and that the further epitheliomatous development followed. The growths from the epidermis are the more characteristic of malignancy.

In conclusion: The clinical course and the histological structure both go to show that the disease has, up to this time, been free from any malignancy, and that we may yet be able to save the patient from the horrors of destructive carcinoma.



**FIG. 8.—Amorphous collections of epithelial cells, seen under higher process of the microscope, around the spaces (a and b).**

34

# INDEX TO VOLUME I.

## (EIGHTH SERIES.)

### A.

Abdominal aorta, aneurism of the, 158.  
 Abscess of the mastoid process, 336.  
*Acarus hominis*, 91.  
 Accommodation in hypermetropia, 323.  
 Acetanilide in headache, 59.  
 Aconite in whooping-cough, 42.  
 Affections of the intrinsic and extrinsic muscles of the eye, 308.  
     symptoms of, 308.  
     treatment of, 308.  
 Albuminuria in typhoid fever, 59.  
 Alcohol causing myocarditis, 111.  
     in tuberculosis, 177.  
 Aloes in coprostasis, 102.  
 Amenorrhœa, sequelæ of, 146.  
 Ammonia and ether mixture in pneumonia, 13.  
     aromatic spirits of, as a carminative, 119.  
     in typhoid fever, 62.  
 Ammonium carbonate in pneumonia, 13.  
 Anæmia and chlorosis, 67.  
     in typhoid fever, 57.  
     the murmur of, 165.  
     the treatment of, 81.  
 Aneurism of the abdominal aorta, 158.  
     the symptoms of, 161.  
 Angina pectoris, 114.  
 Antifebrin in the treatment of scleritis episcleritis, 311.  
 Antimony, tartarized, in labor-pains, 24.  
 Antipyrin in the treatment of scleritis and episcleritis, 311.  
     in whooping-cough, 43.  
 Aortic disease, 164.  
     incompetence, symptoms of, 171.  
     tracing of, 168.  
     regurgitation, 178.  
 Aperients in chlorosis, 74.  
 Apomorphine, the use of, 17.  
 Appendicitis, benzo-naphthol in, 103.  
     morphine in, 19.  
     opium in, 104.  
     potassium chlorate in, 104.  
     silver nitrate in, 104.  
     the preventive treatment of, 98.  
 Aqueduct of Sylvius, lesion of the, 331.  
 Arsenic in aneurism, 162.  
 Arterial sclerosis and cardiac disease, 112.  
     the etiology of, 159.  
 Arthritis complicating osteomyelitis, 253.  
 Asepsis in obstetrics, 84.  
 Asphyxia neonatorum, 52.

Asthenia, cardiac, massage in, 125.  
     *nux vomica* in, 125.  
     Schott baths in, 125.  
     symptoms of, 123.  
     treatment of, 125.  
 Asthenopia from hypermetropia, 323.  
 Atheroma, a contraindication to digitalis, 16.  
     the etiology of, 160.  
 Atropine sulphate in hypermetropia, 323.  
     in cardiac disease, 24.  
     in the treatment of scleritis and episcleritis, 311.  
     in typhoid fever, 62.

### B.

Balsam of Peru in scabies, 96.  
 Baths in scabies, 96.  
 Beer heart, 106.  
 Belladonna in coprostasis, 102.  
     in whooping-cough, 41.  
 Benzo-naphthol in appendicitis, 103.  
 Beta-naphthol in scabies, 96.  
 Bisferiens pulse, 166.  
 Bismuth salicylate in coprostasis, 103.  
     salts in the treatment of acute osteomyelitis, 263.  
 Bland's pills in chlorosis, 82.  
 Blisters in the treatment of spinal irritation, 208.  
 Brandy in pneumonia, 13.  
 Bread, a substitute for, in diabetes, 35.  
 Bromoform in whooping-cough, 43.  
 Bronchitis, chronic, digitalis in, 12.  
 Bronchopneumonia, complicating whooping-cough, 46.

### C.

Cæcum, catarrhal inflammation of the, 101.  
 Caffeine citrate in headache, 59.  
 Calcium chloride in tuberculosis, 177.  
 Camphor in tuberculosis, 177.  
     monobromate in headache, 59.  
 Cancer simulating syphilitic stricture of the rectum, 239.  
 Cannabis indica in tuberculosis, 177.  
 Carbolic acid as an oral disinfectant, 47.  
 Carbuncle, a case of, 13.  
 Cardiac asthenia, 124.  
     collapse, the symptoms of, 110.  
     degeneration in chloroform narcosis, 111.  
     dilatation in chlorosis, 70.  
     disease, the etiology of, 12.  
     the treatment of, 24.



Cardiac dulness in tachycardia, 191.  
 inflammation, 105.  
 manifestations of acute rheumatism, 2.  
 weakness, 123.  
 Caries, mastoid, 335.  
 Castor oil in coprostitis, 102.  
 Cataract, operation for the extraction of, 319.  
   rheumatic or gouty, 316.  
 Caustics in epithelioma, 340.  
 Cephalalgia, acetanilide in, 59.  
   caffeine citrate in, 59.  
   camphor monobromate in, 59.  
   the treatment of, 58.  
 Cerebral rheumatism, the symptoms of, 1.  
 Cheyne-Stokes respiration in general paralysis, 217.  
 Chicago sanitary flour, 35.  
 Chicken-pox, the diagnosis of, 129.  
 Chloro-anæmia, 146.  
 Chloroform as a carminative, 119.  
   in the convulsions of whooping-cough, 47.  
   narcosis, cardiac degeneration in, 111.  
 Chlorosis, cardiac dilatation in, 70.  
   constipation in, 69.  
   food in, 74.  
   hæmoglobin in, 69.  
   iron in, 69, 82.  
   nervous debility in, 70.  
   rest in, 72, 76.  
   the complications of, 69.  
   the etiology of, 69.  
   the treatment of, 67, 76.  
   thrombosis in, 67.  
   tuberculosis following, 68.  
   ulcer of the stomach in, 71.  
 Cholera, the treatment of, 21.  
 Choroiditis and irido-choroiditis, 313.  
 Circular craniectomy for microcephaly, 223.  
   a case of, 223.  
   method of, 224.  
 Cirrhosis of the liver, the treatment of, 12.  
 Cocaine in cataract operations, 321.  
   in epithelioma, 341.  
 Cold baths in the treatment of rheumatism, 1.  
 Colon washing in typhoid fever, 61.  
 Conjunctivitis, rheumatic and gouty, 308.  
   a case of, 308.  
   diagnosis of, 308.  
   extravasation in, 308.  
   pannus following, 308.  
   sympathetic, 324.  
 Constipation and chlorosis, 69.  
 Contagiousness of scabies, 93.  
 Coprostitis, belladonna in, 102.  
   benzo-naphthol in, 103.  
   bismuth salicylate in, 103.  
   castor oil in, 103.  
   hyoscyamus in, 102.  
   intestinal antiseptics in, 103.  
   in typhoid fever, 99.  
   jalap in, 102.  
   mercurial ointment in, 103.  
   morphine in, 103.  
   the symptoms of, 100.  
   treatment of, 102.  
 Coronary arteries, embolism of the, 113.  
 Counter-irritation in whooping-cough, 47.  
 Creosote in the treatment of tuberculosis, 186.  
   in whooping-cough, 45.

Croton oil in whooping-cough, 47.  
 Curettage in endometritis, 51.  
   in puerperal sepsis, 87.  
 Cystitis, a case of, 13.

## D.

Deformity following tubercular abscess of hip-joint, 225.  
 Delirium in rheumatism, 2.  
   opium in, 17.  
 Diabetes, the treatment of, 35.  
 Diarrhoea, the treatment of, 21.  
 Diet in diabetes, 35.  
   in typhoid fever, 59.  
   of gastrostasis, 128.  
 Digitalis as a diuretic, 8.  
   effect on the circulation of, 12.  
   four stages of the action of, 13.  
   in myocarditis, 115.  
   in tachycardia, 190.  
   in whooping-cough, 42.  
   the infusion of, 10.  
   the preparations of, 9.  
 Digitonin, the action of, 9.  
 Dilatation of the os uteri in placenta prævia, 55.  
 Diphtheria, cardiac degeneration in, 107.  
 Dissection of cataract, 321.  
 Diuretic action of digitalis, 9.  
 Duodenal ulceration, a case of, 150.  
 Dyspepsia, a case of, 99.  
   and chlorosis, 76.  
   chronic, 126, 151.  
   opium in, 24.  
   the treatment of, 35.  
 Dyspnoea, cardiac, 188.

## E.

Eberth bacillus causing osteomyelitis, 257.  
 Eczema of the eyelid, rheumatic and gouty, 309.  
   the diagnosis of, 93.  
 Embolism in infectious fevers, 109.  
   of the coronary arteries, 113.  
 Emetics in whooping-cough, 45.  
 Emphysema, digitalis in, 12.  
 Empyema of the mastoid process, 336.  
 Endocarditis, a case of, 144.  
   chronic, 110.  
   rheumatic, 2, 108.  
   the symptoms of, 145.  
   the treatment of, 145.  
   ulcerative, 118.  
 Endometritis following placenta prævia, 51.  
 Enemata, nutritive, in typhoid fever, 61.  
 Enteritis and appendicitis, 99.  
   the treatment of, 23.  
 Epistaxis in whooping-cough, 40.  
 Epithelioma of the vulva, 286.  
   a case of, 286.  
   diagnosis of, 286.  
   diseases simulating, 287.  
   duration of, 287.  
   prognosis of, 288.  
   treatment of, 288.  
   recurrent, 340.  
   the pathology of, 341.

Ergot in the treatment of uterine hemorrhage, 298.  
 Eruption of varicella, 132.  
 Ether in the convulsions of whooping-cough, 47.  
 Eucalyptol in the treatment of acute osteomyelitis, 262.  
 Eucalyptus in whooping-cough, 45.  
 Eustachian tube, inflammation of the, 333.  
 Exudation of rheumatism, micrococci in, 5.  
 Eye-diseases, sympathetic, 324.

F.

Fæces, chemical composition of, in coprostasis, 100.  
 Femoral artery, diastolic murmur in, 173.  
     tubular aneurism of, 235.  
 Fever, the use of opium in, 20.  
 Flour, sanitary, for certain dyspeptics and diabetics, 35.  
 Fetus, dangers to the, in placenta prævia, 51.  
 Furuncle of the external auditory meatus, 337.

G.

Gastralgia in appendicitis, 99.  
     in chlorosis, 71.  
 Gastroctasis, 126.  
 Gastric hyperasthenia, 99.  
     juice, chemical composition of, in appendicitis, 99.  
     ulcer in chlorosis, 67.  
 Gastro-enterostomy in pyloric obstruction, 156.  
 General paralysis as met with in hospital practice, 209.  
     case of, 211.  
     diagnosis of, 213.  
     symptoms of, 212.  
     treatment of, 218.  
 Glands, ductless, the secretion of, 20.  
 Goitre, a case of, 221.  
     operation for, 222.  
     removal of, 222.  
     exophthalmic, 190.  
 Gouty iritis, 311.  
 Green soap in scabies, 96.

H.

Hæmoglobin in chlorosis, 69.  
 Heart, aortic disease of the, 164.  
     inflammation of the muscle of the, 107.  
     nerves of the, 189.  
     valvular diseases of the, 106.  
 Hemorrhage, cerebral, in whooping-cough, 40.  
     in placenta prævia, 49.  
     intestinal, in typhoid fever, 57.  
     meningeal, in whooping-cough, 40.  
     morphine in, 23.  
     uterine, 295.  
 Hernia, umbilical, 219.  
 Hip-joint, rheumatism of the, 7.  
     tubercular abscess of the, 225.  
 Histology of epithelioma, 345.  
 Homatropine in hypermetropia, 323.  
 Hydrargyrum oxidum flavum in furuncle, 337.  
 Hydrochloric acid in the gastric juice, 155.  
 Hyoscine in typhoid fever, 61.

Hyoseyamus in coprostasis, 102.  
     in tuberculosis, 177.  
 Hypermetropia, a case of, 323.  
 Hyperpyrexia in typhoid fever, 63.  
 Hysterectomy in septic infection, 89.

I.

Infant, danger to the, in placenta prævia, 51.  
 Infectious diseases and the puerperium, 85.  
     fevers, sudden death in, 109.  
 Influenza and tuberculosis, 175.  
 Intestinal antiseptics in appendicitis, 103.  
 Iodide of potassium in the treatment of scleritis and episcleritis, 311.  
 Iodine in whooping-cough, 47.  
 Iodoform in puerperal infection, 87.  
     in the treatment of acute osteomyelitis, 263.  
 Irido-cyclitis, 325.  
 Iritis, rheumatic or gouty, 311.  
     syphilitic, 328.  
 Irodotomy, 322.  
 Iron in chlorosis, 69.  
     the carbonate of, in chlorosis, 82.  
 Irritation of the spine, 193.

J.

Jalap, resin of, in coprostasis, 102.

K.

Keratitis punctata, 325.  
     rheumatic, 309.  
     sympathetic, 321.  
 Koumiss in typhoid fever, 62.

L.

Labor-pains, the relief of, 24.  
 Lacerations and cicatrices of the vagina, 281.  
     a case of, 281.  
     symptoms of, 281.  
     treatment of, 281.  
 Laparotomy for dilated stomach, 150.  
     for general peritonitis, 103.  
     for ulceration of the pylorus, 149.  
 Larva of the acarus hominis, 92.  
 Larynx, condition of, in tuberculosis, 177.  
 Laudanum, the action of, 17.  
 Lavage of the stomach, 127.  
 Leeches in the treatment of spinal irritation, 208.  
 Lesions of scabies, 92.  
 Locomotor ataxia, the symptoms of, 331.

M.

Malaria, the diagnosis of, 58.  
 Malarial fever and splenic enlargement, 136.  
 Massage in the treatment of cardiac weakness, 125.  
     in the treatment of varicose veins, 25, 31.  
 Mastoid caries, 335.  
 Mastoiditis following whooping-cough, 338.  
 Mediastinum, tumor of the, the symptoms of, 39.  
 Meningitis in typhoid fever, 57, 63.  
 Menthol as a carminative, 119.  
 Mercurial ointment in coprostasis, 103.

**Mercuric bichloride** in puerperal infection, 87.  
**Mercury** in aneurism, 162.  
     yellow oxide of, in furunculosis, 337.  
**Microcephaly**, a case of, 223.  
     operation for, 224.  
     symptoms of, 223.  
**Mineral contents of the faeces**, 100.  
     waters in whooping-cough, 47.  
**Mitral incompetence**, 173.  
**Morphia** in appendicitis, 103.  
     sudden death from, 113.  
     the discovery of, 17.  
**Motor oculi paralysis**, 329.  
**Movable kidney, nephrorrhaphy** for, 231.  
**Mucocomembranous enteritis**, 101.  
**Mucous membrane** in whooping-cough, 40.  
**Murmur of aortic regurgitation**, 179.  
     systolic, in aortic disease, 164.  
**Myocarditis**, 105.  
     from alcohol, 111.  
     from syphilis, 110.  
**Myomalacia cordis, acute**, 111.

## N.

**Naphthol** in appendicitis, 103.  
**Naso-pharyngitis, acute**, 333.  
**Nephritis, acute, digitalis** in, 11.  
**Nephrorrhaphy** for movable kidney, 231.  
**Nervous debility** in chlorosis, 70.  
**Nuclein injections** in the treatment of tubercular joint troubles, 226.  
     the action of, 20.  
**Neurasthenia and chlorosis**, 77.  
**Neuroretinitis**, 326.  
**Nitroglycerin** in typhoid fever, 62.  
     the diuretic effect of, 15.  
**Nux vomica** in cardiac weakness, 125.  
     in tuberculosis, 177.

## O.

**Oedema, cardiac**, 188.  
**Occipital neuralgia** in typhoid fever, 58.  
**Opacities** in the vitreous, rheumatic, 316.  
**Operation** for extraction of cataract, 319.  
     for microcephaly, 224.  
     for syphilitic stricture of the rectum, 243.  
     for the removal of a left-sided goitre, 222.  
     for umbilical hernia, 220.  
**Opium and bismuth** in diarrhoea, 21.  
     in appendicitis, 104.  
     in cholera, 21.  
     in typhoid fever, 62.  
     the use of, 17.  
**Optic nerve, sympathetic inflammation of the**, 326.  
     neuritis, rheumatic, 314.  
     cases of, 314.  
     treatment of, 314.  
**Orbit, periostitis of the**, 306.  
     a case of, 306.  
     symptoms of, 306.  
     treatment of, 306.  
**Osteomyelitis, in young children, acute**, 249.  
     a case of, 259.  
     complications of, 252, 258.  
     diagnosis of, 253, 259.  
     diarrhoea a symptom of, 249.  
     fluctuation a symptom of, 252.

**Osteomyelitis in young children, microbes** causing, 255.  
     most frequent seat of, 253.  
     pain a symptom of, 250.  
     swelling a symptom of, 251.  
     treatment of, 260.  
**Otitis media, acute**, 333.  
**Ovaritis after placenta prævia**, 51.  
**Oxytuberculin, the value of**, 186.

## P.

**Pain, the etiology of**, 18.  
**Pancreas, ulceration of the**, 150.  
**Paralysis agitans, a case of**, 331.  
     of the oculo-motor nerve, 329.  
**Parasite of scabies**, 91.  
**Pathology of general paralysis**, 217.  
**Pelvic peritonitis and fibroid tumor**, 277.  
     a case of, 277.  
     diagnosis of, 278.  
     treatment of, 277.  
**Perforation of the stomach**, 118.  
**Pericarditis** in rheumatism, 2.  
**Periostitis of the orbit**, 306.  
**Peritonitis, acute, perforative**, 116.  
     following gastric ulcer, 122.  
     the treatment of, 23.  
**Peroxide of hydrogen** in furunculosis, 337.  
**Pharyngitis, specific**, 177.  
**Pharynx, infection of the, in rheumatism**, 6.  
**Phenol** in the treatment of acute osteomyelitis, 262.  
**Phlegmasia alba dolens** in placenta prævia, 51.  
**Phthisis bulbi**, 326.  
     fibroid, 141.  
     the diagnosis of, 142.  
     the treatment of, 144.  
**Pills, the non-absorption of**, 22.  
**Pilocarpine, in the treatment of scleritis and episcleritis**, 311.  
**Placenta prævia**, 264.  
     a case of, 264, 265.  
     ergot in, 268.  
     extraction of foetus in, 268.  
     plugging in, 268.  
     pulse-rate in, 267.  
     the diagnosis of, 265.  
     treatment of, 48, 264.  
**Pleurisy, tubercular**, 180.  
**Pneumococcus** causing osteomyelitis, 257.  
**Pneumograph, Marey's, the use of**, 39.  
**Pneumonia, catarrhal, a case of**, 60.  
     double, 118.  
     following carbuncle, 18.  
     sudden death in, 109.  
**Pneumonitis, the treatment of**, 23.  
**Popliteal aneurism**, 162.  
**Potassium chlorate** in appendicitis, 104.  
     iodide in aneurism, 162.  
     in iritis, 328.  
     in specific pharyngitis, 178.  
**Pregnant uterus, retroversion of the**, 284.  
**Preventive treatment of appendicitis**, 93.  
**Puerperal pulmonary thrombosis**, 51.  
     insanity after placenta prævia, 51.  
     sepsis, antiseptic treatment of, 84.  
     curettage in, 87.  
     iodoform in, 87.  
     hysterectomy in, 89.

Puerperal sepsis, mercuric bichloride in, 87.  
the etiology of, 85.  
the treatment of, 85.  
Pulmonary engorgement, digitalis in, 12.  
tuberculosis, 175.  
Pulse, rapidity of, in tuberculosis, 176.  
waves in aortic stenosis, 166.  
Pupil, inequality of, in locomotor ataxia, 332.  
Purgatives in appendicitis, 104.  
Purpura in whooping-cough, 40.  
Pyloroplasty in gastric dilatation, 156.  
the statistics of, 157.  
Pylorus, stenosis of the, 127, 151.  
ulceration of the, 146.

R.

Rapid dilatation of syphilitic rectal strictures, 243.  
Rectum, syphilitic stricture of the, 239.  
Reflexes in general paralysis, 213.  
Regurgitation, aortic, 178.  
Resection in acute osteomyelitis, 262.  
Resorcin as a disinfectant for the mouth, 46.  
Retention of urine, urethrotomy for, 227.  
Retinitis hemorrhagica, 315.  
cases of, 315.  
symptoms of, 316.  
Retroflexion and prolapsed ovary, 278.  
a case of, 279.  
diagnosis of, 279.  
treatment of, 280.  
Retroposition of the uterus, 289.  
a case of, 289.  
causes of, 289.  
symptoms of, 289.  
treatment of, 290.  
Retroversion of the pregnant uterus, 284.  
a case of, 284.  
pessaries in, 285.  
symptoms of, 284.  
treatment of, 284.  
Rheumatic and gouty diatheses and their relation to diseases of the eye, 303.  
endocarditis, 108.  
iritis, 311.  
keratitis, 309.  
a case of, 309.  
diagnosis of, 309.  
iritis complicating, 309.  
treatment of, 309.  
Rheumatism, acute articular, complications of, 4.  
and arterio-sclerosis, 159.  
cerebral, symptoms of, 1.  
inflammatory, treatment of, 1.  
the bacteriology of, 6.  
the etiology of, 6.  
the infectious nature of, 6.  
the micro-organism of, 4.  
the statistics of cardio-pulmonary manifestations in, 3.  
the treatment of the visceral manifestations of, 1.  
Rhinitis in whooping-cough, 47.  
Rupture of a tubular aneurism of the femoral artery, 235.

S.

Salicylate of sodium in rheumatism, 1.  
Salicylates in the treatment of scleritis and episcleritis, 311.  
Salines in peritonitis, 23.  
Salol in typhoid fever, 62.  
Scabies, *acarus hominis* of, 91.  
balsam of Peru in, 96.  
baths in, 96.  
beta-naphthol in, 96.  
contagiousness of, 93.  
green soap in, 96.  
lesions of, 92.  
sulphur in, 96.  
styrax in, 96.  
the treatment of, 91.  
Schott baths in cardiac weakness, 125.  
Scleritis and episcleritis, 310.  
symptoms of, 310.  
treatment of, 311.  
Scleritis, sympathetic, 324.  
Sensory function in general paralysis, 213.  
Sepsis, the avoidance of, during the puerperium, 85.  
Septic infection in placenta prævia, 50.  
Shock, opium in the treatment of, 19.  
Silver nitrate in appendicitis, 104.  
in epithelioma, 341.  
in stomatitis, 47.  
Skin-grafting after epithelioma, 341.  
Sleep from opium, 17.  
Small-pox, the eruption of, 130.  
Sodium arsenate in splenic enlargement, 140.  
benzoate in whooping-cough, 45.  
salicylate, contraindications to the use of, 1.  
Spinal irritation, 193.  
cases of, 195.  
causes of, 194.  
diagnosis of, 194, 195.  
symptoms of, 194.  
treatment of, 195.  
Spleen, enlargements of the, 135.  
Sphygmographic tracings of the radial pulse, 166.  
Staphylococcus aureus in rheumatism, 4.  
causing osteomyelitis, 255.  
Stenosis, aortic, 165.  
Stomach contents, examination of the, 127.  
digestion, 154.  
dilatation of the, 72, 149, 151.  
tests, 154.  
ulceration of the, 147.  
causing peritonitis, 116.  
in chlorosis, 71.  
the etiology of, 147.  
Stomatitis in whooping-cough, 47.  
Streptococci serum in septic absorption, 271.  
Streptococcus causing osteomyelitis, 255.  
Strophanthus, the action of, 12.  
Strychnine in appendicitis, 19.  
in pneumonia, 13.  
in typhoid fever, 61.  
the action of, 19.  
Styrax in scabies, 96.  
Sulphur in scabies, 96.  
Syncope in whooping-cough, the treatment of, 47.  
Synecchia of iritis, 328.

**Syphilis and arterio-sclerosis, 169.**

causing paralysis, 331.  
in pyloric ulceration, 146.

**Syphilitic iritis, 328.**

stricture of the rectum, 239.  
a case of, 239.  
causes of, 239.  
diagnosis of, 241.  
medicinal treatment of, 245.  
operation for, 248.  
rapid dilatation of, 243.  
symptoms of, 240.

**Systolic murmur in aortic disease, 164.****T.****Tachycardia, cardiac dulness in, 191.**

digitalis in, 190.  
the symptoms of, 187.  
the treatment of, 190.

**Tenonitis, 307.****Terpinol in whooping-cough, 45.**

Thebaine, the antispasmodic action of, 17.

**Thrombosis in chlorosis, 67, 70.****Tobacco causing myocarditis, 112.****Trachelorrhaphy, the after-treatment of, 282.****Treatment of acute inflammatory rheumatism, 1.**

osteomyelitis in children, 260.  
antisepsis in the, 262.  
bismuth salts in the, 263.  
ourettement in the, 262.  
drainage in the, 262.  
eucalyptol in the, 262.  
iodoform in the, 263.  
phenol in the, 262.  
resection in the, 262.  
thymol in the, 263.  
trephining in the, 260.

of affections of eye muscles, 308.  
inunctions of oil in the, 308.  
massage in the, 308.  
salicylates in the, 308.

of anemia, 81.

of aneurism, 162.

of cardiac disease, 24.

of chlorosis, 67.

of cholera, 21.

of choroiditis and acute glaucoma, 314.

eserine in the, 314.

iridectomy in the, 314.

pilocarpine in the, 314.

salicylates in the, 314.

of convulsions in whooping-cough, 47.

of coprostitis, 102.

of diabetes, 35.

of diarrhoea, 21.

of dilatation of the stomach, 149, 151, 155.

of dysentery, 21.

of dyspepsia, 24, 35.

of epithelioma, 341.

of excessive mobility of the uterus, 277.

pessaries in the, 277.

of furuncle of the ear, 337.

of general paralysis, 218.

bromide of potassium in the, 218.

cerebrin in the, 218.

iodide of potassium in the, 218.

thyroid extract in the, 218.

**Treatment of general paralysis, trephining in the, 218.**

of gouty and rheumatic iritis, 312.

atropine in the, 312.

cocaine in the, 312.

colobium in the, 313.

leeches in the, 313.

morphine in the, 313.

oil of gaultheria in the, 312.

salicylates in the, 313.

of headache, 58.

of hemorrhages, 23.

of labor-pains, 24.

of mastoid abscess, 339.

of motor oculi paralysis, 331.

of myocarditis, 115.

of naso-pharyngeal catarrh, 334.

of optic neuritis, 315.

leeches in the, 315.

salicylates in the, 315.

of paralysis of the oculo-motor nerve, 331.

of periorbitis of the orbit, 306.

of peritonitis, 23.

of placenta prævia, 48, 53, 264.

of popliteal aneurism, 162.

of pneumonia, 13.

of pneumonitis, 23.

of puerperal sepsis, 83.

of retroflexion and prolapsed ovary, 280.

best position for the, 280.

operation in the, 281.

pessaries for the, 280.

of retroposition of the uterus, 290.

massage in the, 290.

operative, 291.

tampons in the, 290.

of rheumatic keratitis, 309.

of scabies, 91.

of scleritis and episcleritis, 311.

antifebrin in the, 311.

antipyrin in the, 311.

atropine in the, 311.

cocaine in the, 311.

iodide of potassium in the, 311.

pilocarpine in the, 311.

salicylates in the, 311.

of specific pharyngitis, 178.

of spinal irritation, 196.

blisters in the, 208.

leeches in the, 208.

of splenic enlargement, 139.

of syncope in whooping-cough, 47.

of the puerperium, 85.

of trachelorrhaphy, 282.

carbolyzed douches in the, 282.

duration of the, 282.

tampons in the, 283.

of tubercular abscess of hip-joint, 225.

nuclein injections in the, 226.

of tuberculosis, 144, 177.

of typhoid fever, 57.

of ulceration of the pylorus, 149.

of uterine hemorrhage, 298.

ourettement in the, 297.

ergot in the, 298.

of varicose veins, 25.

of whooping-cough, 38.

Trephining in acute osteomyelitis, 260.

Tricuspid incompetence, 178.

- Tubercular abscess of hip-joint, 225.  
     a case of, 225.  
     deformity following, 225.  
     treatment of, 225.  
     mastoiditis, 338.  
     pleurisy, 180.  
         the symptoms of, 181.  
         the treatment of, 186.  
 Tuberculosis following chlorosis, 68.  
     pulmonary, 175.  
         alcohol in, 177.  
         calcium chloride in, 177.  
         camphor in, 177.  
         cannabis indica in, 177.  
         creosote in, 186.  
         hyoscyamus in, 177.  
         nux vomica in, 177.  
         the symptoms of, 173.  
         the treatment of, 177.  
 Tubular aneurism of the femoral artery, 235.  
     a case of, 235.  
     cause of, 235.  
     operation for, 236.  
     rupture of, 235.  
 Tumor of the spleen, 137.  
 Tympanites in typhoid fever, 61.  
 Typhoid fever, albuminuria in, 59.  
     aromatic spirits of ammonia in, 62.  
     atropine in, 62.  
     colon washing in, 61.  
     diet in, 59.  
     enemata, nutritive, in, 61.  
     hyoscyne in, 61.  
     hyperpyrexia in, 63.  
     koumiss in, 62.  
     meningitis in, 57, 63.  
     nitroglycerin in, 62.  
     opium in, 60.  
     pneumonia in, 60.  
     salol in, 62.  
     strychnine in, 61.  
     sudden death in, 109.  
     treatment of, 62.  
     tympanites in, 61.  
     Widall's test for, 61.

U.

- Ulcer, congested, the pathology of, 28.  
     of the stomach, fatal, 119.  
 Ulceration, duodenal, 150.  
     of the pancreas, 150.  
     of the pylorus, 146.  
     of the stomach in chlorosis, 71.  
 Ulcers, congested, 25.  
 Umbilical hernia, 219.

- Umbilical hernia, a case of, 219.  
     operation for, 220.  
 Urethrotomy for retention of urine, 227.  
     after-treatment of, 230.  
     method of, 229.  
 Urine, the secretion of, under the influence of digitalis, 14.  
 Uterine hemorrhage, 295.  
     causes of, 295.  
     digital examination in, 296.  
     treatment of, 298.  
 Uterus, excessive mobility of the, 276.  
     a case of, 276.  
     the diagnosis of, 276.  
     the symptoms of, 277.  
     the treatment of, 277.  
     infection of the, 87.  
     retroposition of the, 289.

V.

- Vaccination and variola, 133.  
 Vagina, lacerations and cicatrices of, 281.  
 Valerian in whooping-cough, 42.  
 Varicella, the diagnosis of, 129.  
 Varicose veins, the etiology of, 26.  
     the pathological sequelæ of, 27.  
     the treatment of, 25.  
 Venectomy for varicose veins, 25.  
 Ventilation, the importance of, in whooping-cough, 45.  
 Vulva, epithelioma of the, 288.

W.

- Whooping-cough, aconite in, 42.  
     antipyrin in, 43.  
     belladonna in, 41.  
     bromoforn in, 43.  
     creosote in, 45.  
     digitalis in, 42.  
     epistaxis in, 40.  
     eucalyptus in, 45.  
     hemorrhage, cerebral, in, 40.  
     meningeal, in, 40.  
     mucous membranes in, 40.  
     purpura in, 40.  
     sodium benzoate in, 45.  
     terpinol in, 45.  
     the sequelæ of, 338.  
     the symptoms of, 40.  
     the treatment of, 38.  
     turpentine in, 45.  
     valerian in, 42.  
 Widall's typhoid fever test, 61.

END OF VOLUME I.

UNIV. OF MICHIGAN,

FEB 26 1922















3 9015 07037 1128

